THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888.

No. 7. FEBRUARY 15, 1947 Vol. 59.

CENTRAL VETERINARY SOCIETY
"Clinical Forum" Meeting

At the General Meeting of the Society held on Wednesday, January 3rd, 1946, there was a variation of the usual type of discussion.

Members of the staff of the Royal Veterinary College—Dr. Amoroso, Professor Bosworth, Professor Burrow, Professor Edholm, Mr. Muir, Dr. Oldham and Professor Wooldridge—formed a group for the discussion of specific questions chosen from those submitted beforehand by the Fellows and passed on to the group in order that they could give considered answers. After each question was answered the members of the group discussed it, and after all the questions had been dealt with the discussion became open to all present. This proved to be a most stimulating meeting and it is hoped that, in some form, it may be possible to hold a similar one in the near future.

We reproduce below the questions and the discussions on them.

Site of Lesions in Dogs Showing C.N.S. Symptoms

Is it possible in the present state of expert veterinary knowledge to say which part of the brain, cord or their covering membranes is affected when dogs show symptoms affecting the central nervous system?

Professor T. J. Bosworth: Emphasis here should be on the word expert and the answer might well have been entrusted to someone with considerable clinical experience in interpreting nervous symptoms in dogs, or alternatively to a neuro-physiologist with some exact knowledge of the effects of lesions experimentally produced in the different parts of the central nervous system. Diseases of the brain and cord are not at all uncommon and while in some cases their diagnosis and differential diagnosis present considerable difficulties I think it can be said in answer to this question that the symptoms shown often indicate the probable site of the lesion, but do not necessarily afford a clue as to its actual cause. So far as the interpretation of nervous symptoms is concerned the veterinarian, for various reasons, does not appear to be in as good a position as that enjoyed by the human neurologist though since the dog is frequently the subject of experiment by physiologists there may be more detailed information available regarding the functions of the different parts of its brain than is generally recognized. Some of the existing possibilities in regard to diagnosis are illustrated in a paper by Schlotthauer* who studied the correlation of symptoms with lesions in a series of 78 affected dogs and in all cases but four was able to find some lesions in the central nervous system to account for the symptoms observed. About two-thirds of the animals showed some form of encephalitis. Disease of the cerebrum was manifested by disturbances in psychic behaviour, impaired intelligence, inco-ordination of movement and epileptiform convulsions. Chorea was almost always due to a focal lesion affecting the cerebrum, while continuous walking in a circle and a tendency to unnatural gait were commonly related to encephalitis of the disseminated type. Where the cerebellum alone was affected ataxia without evidence of psychic upset or paralysis was usually seen. Signs of clumsy gait with progressive loss of intelligence and impaired vision due to papilloedema were prominent in cases of hydrocephalus in which condition convulsions were never observed. Though paralysis if the legs might arise from lesions involving the motor tracts in the brain, cases of it in the dog are almost always due to a spinal lesion. These are a few illustrations which tend to show that careful detection and interpretation of the symptoms will in many cases indicate the part of the brain which is affected.

Discussion

Mr. W. R. Muir: On the question of causes, I would like to draw attention to the fact that diseases of the central nervous system can arise from dietary deficiencies such as lack of vitamin A and certain factors of the vitamin B complex. For example, Sir Edward Mellanby has shown that hydrocephalus can develop in young vitamin A deficient puppies on account of hypertrophy of the bones of the posterior fossa. The space available for the cerebellum and brain stem is thereby restricted although the whole brain continues to grow. Pressure is thus exerted on the brain and the fourth ventricle becomes wholly or partially occluded between the cerebellum and medulla thereby preventing the passage of cerebrospinal fluid to the subarachnoid spaces in the posterior fossa and spinal cord. Since fluid continues to be secreted by the choroid plexus of the third and lateral ventricles a condition of internal hydrocephalus results. I do not wish to suggest that all cases of hydrocephalus are due to vitamin A deficiency. Bacteriologists may well be able to point to other causes of hydrocephalus but I would like to draw attention to the fact that vitamin A deficiency can be at least one cause of this condition. Papilloedema and inco-ordination of movement were also observed by Mellanby in his vitamin A deficient dogs. With regard to vitamin B, the effects of B_1 deficiency on brain function are well known. Riboflavin deficiency in dogs can be accompanied by nerve cell degeneration. In advanced cases of riboflavin deficiency mild patchy degeneration of the spinal cord has been observed. A spastic gait has been reported to be one of the symptoms shown by affected dogs. Acute riboflavin deficiency in dogs is marked by sudden collapse and death. Yellow atrophy of the liver is a prominent *post-mortem* feature.

Professor Bosworth: Does vitamin B deficiency occur in dogs in natural circumstances or is it merely an experimental disease?

Mr. Muir: Nicotinic acid (anti-blacktongue factor) deficiency definitely does, but whether riboflavin deficiency occurs I am not sure. I have seen several dogs in the hospital at Camden Town which appeared to be cases of riboflavin deficiency but I have been unable to arrive at a definite decision. Vitamin deficiencies are usually multiple, not single. The average dog's diet now, as a result of food restrictions, is mostly potatoes, gravy and white bread which is deficient in riboflavin, nicotinic acid and a number of other factors for dogs.

Professor Bosworth: During the war period there have been some rather obscure cases of inco-ordination of movement in dogs kept on a very restricted diet and this has naturally led to speculation as to whether they might be due to some vitamin or other deficiency. My colleagues and I have in some cases kept these animals under observation and have fed them for a period on what we believed to be a fully adequate diet, but so far we have not been able to effect a cure.

Mr. Muir: Some of the changes are irreversible and therefore a cure cannot always be expected. That fact has been proved experimentally. The point is that when the veterinary surgeon sees a dog suffering from a deficiency disease, the condition has usually progressed too far.

Professor Bosworth: In regard to vitamin A deficiency, are cases of it showing marked degeneration of the cranial nerves, as produced by Mellanby, likely to arise in practice?

Mr. Muir: If vitamin A deficiency is to be responsible for nerve degeneration, then the deficiency state must commence in the puppy or young growing dog. The possibility of vitamin A deficiency occurring in the dog under ordinary conditions of domestication is extremely likely because biscuit and meat diets are deficient in vitamin A.

Professor G. H. Wooldridge: In some instances symptoms do point to the seat of the lesion in the nervous system but not by any means in all. There are several examples, particularly in those dogs with long backs such as Dachshunds, Pekes, etc., with paralysis of the hind legs due to lumbar trouble—first there is a condition of lumbar hyperaesthesia and later the painful phase subsides and loss of motor power supervenes. Quite a number of radiographers including Mr. Kirk have demonstrated in a number of instances some bony or calcareous deposits about the foramina of the lumbar vertebrae, causing nervous derangement. This is undoubtedly the seat of the trouble. You get the painful symptoms first by irritation of the nerves, and as pain subsides motor power is impaired. After a time in many cases accommodation takes place and both functions are restored though recurrences often occur. In these examples it is possible to locate the seat of the trouble within one or two of the lumbar vertebrae. In other instances it is possible to locate lesions in the brain. In one particular case I was able from the symptoms to locate the area involved and arrive at a diagnosis. It was a fox terrier, male, becoming rather fat and lethargic in its mannerism and there was

* *J.A.V.M.A.* (1940.) **97.** 233.

marked softening and atrophy of the testes and a shrinking of the penis. Later the dog made wide circles to the left and resented attempts to touch its head. My diagnosis was that in all probability there was a growth affecting the pituitary body and more extensive on the left side than on the right. I advised destruction and the dog was destroyed. A *post-mortem* examination was made by Dr. White and I was gratified to find that my diagnosis was entirely correct. That was an instance in which the actual symptoms did lead to the correct diagnosis of the seat of the lesion.* In ruminants you have parasitic cysts and you can locate more or less the seat of the lesion. In the case of sheep (gid) the softening of the bone enables you to locate it. In larger ruminants, also susceptible to the same parasite, the softening of the bone is not transferred to the frontal bone because of the frontal space in between. In Ireland it is fairly common for veterinary surgeons to diagnose the seat of the lesion, solely from the symptoms, to operate and definitely get down to the parasite and remove it. That parasitic trouble does not occur in the dog, but it does suggest that a similar symptom would serve to locate the seat of the lesion, wherever it might be.

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Staphylococcal Infection in Dogs

To what extent may a primary staphylococcus infection be implicated in dogs with chronic ill-health and slight widespread pustular dermatitis (minus Demodex folliculorum*) showing tonsil swab positive for staphylococci?*

Professor Bosworth: It is assumed that the question is meant to ask whether the presence of staphylococci in the tonsils may be held responsible for chronic ill-health associated with mild widespread pustular dermatitis. A somewhat similar question appeared not many weeks ago in the "Queries and Answers" column of *The Veterinary Record*. I more or less agreed with the answer given there except perhaps as to the frequency with which pathogenic staphylococci can be isolated from dogs either in health or otherwise. Experience in the College would suggest that they are not nearly so common in tonsil swabs as streptococci but they are occasionally met with. Haemolytic strains have been isolated from throat swabs from dogs with swollen throat and neck lymph glands, from a throat swab of a dog with furunculosis but also occasionally from throat swabs in cases in which it seemed unlikely that the tonsil was playing any part in the disease noted. I should say that no adequate survey of the incidence of this organism in dogs in health and disease has yet been made, and this question cannot be answered with any degree of certainty. It would be premature to attach significance to the mere presence of staphylococci in the tonsil but unwise to disregard them when they occur in an obviously septic focus accompanied by involvement of the neighbouring lymph glands.

Respiratory Stimulants

What are the most powerful respiratory stimulants, and why is it that in so many cases of respiratory failure the injection of coramine, methedrine, strychnine, and even picrotoxin, seems to exert absolutely no effect?

Professor H. Burrow: The most powerful respiratory stimulant is carbon dioxide and it may be administered by inhalation in 7 per cent. concentration in oxygen. Where respiration has become arrested, artificial respiration must, of course, be resorted to in order to effect its administration. Other powerful respiratory stimulants are the reflex stimulation *via* sensory nerves produced by the application of cold water to the body surface: the reflex stimulation caused by the inflow of cold air into the trachea, and a similar stimulation resulting from manual agitation of the tongue.

Drugs which are stimulants to the central nervous system, such as strychnine, coramine (nikethamide), picrotoxin, in so doing also cause rise in blood pressure *via* the vasoconstrictors, and are not so much selective stimulants of respiration as general stimulants of the central nervous system.

If the central nervous system has become so depressed by anaesthetics, poisons, toxins or shock that it "has lost its power of tone-recovery" then no respiratory stimulant is likely to be of value.

Individual animals must vary in the tolerance possessed by their central nervous systems to depressant agents and, for that reason, the use of anaesthetics must always be fraught with some element of danger.

Discussion

Professor O. G. Edholm: Of the drugs mentioned in the question, coramine is the only one which has any specific action on the respiratory centre.

Professor Wooldridge: In my experience the most rapid and effective respiratory stimulant is a full medicinal dose of prussic acid. It is of the greatest advantage to use it in cases of cessation of breathing in chloroform narcosis. I would never dream of using chloroform unless I had prussic acid available to administer on the back of the patient's tongue (a full medicinal dose) as soon as respiratory disturbances arose. In some cases respiratory restoration is remarkably quick. Apart from prussic acid, I think coramine is perhaps the next best respiratory stimulant.

Mr. Muir: It is very interesting to learn from Professor Wooldridge that prussic acid, which is such a powerful inhibitor of respiratory enzymes, is also a most effective respiratory stimulant when given in medicinal doses. I would like to suggest, as a possible mechanism for this stimulatory action, that prussic acid even in non-toxic doses still inhibits the respiratory enzymes (cytochrome oxidase, etc.) and thereby temporarily creates, in effect, a state of anoxia which is responsible, in turn, for the stimulation of the respiratory centre.

Professor Edholm: I do not think it works in that way. Probably the action of the prussic acid is that of a direct stimulant to the respiratory centre.

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Placental Membranes

Why can the placental membranes be left in a cow for several days without harm, whereas in the mare such is not the case?

Professor H. Burrow: In not every case can placental membranes be left in a cow for several days without harm, but in many instances the statement is true, whereas in the mare, only on rare occasions can the membranes remain for so long as 24 hours without giving rise to acute metritis and/or laminitis.

The reasons are threefold: firstly, the placentation in the mare is diffuse, intimate and most conducive to absorption from the disintegrating and infective foetal membranes into the blood stream of the mare of bacterial toxins and toxic products of protein-lysis. Secondly, horses in general and mares in particular show a much poorer resistance to products of sepsis than do bovines, *e.g.*, pneumonias in horses so often lead to fatal septicaemia while in bovines it is not so frequent. Thirdly, horses are much more sensitive to the action of broken-down proteins than are cattle; this sensitivity is demonstrated by the occurrence of laminitis, and possibly purpura in horses under circumstances remote from the puerperium as well as at that critical time.

Discussion

Dr. Amoroso: I cannot agree that in the mare there is a greater intimacy between the foetal and maternal blood streams. I do, however, agree that there may be a greater effective surface area of contact between the foetal membranes and the maternal uterus in the mare. In the cow, on the other hand, the process of toxic absorption may be restricted because of the separate caruncular areas.

Regarding the suggestion that the mare tends to suffer more from the retention of the membranes than the cow, this may possibly be correlated with the fact that the placenta of the mare is poor in choline whereas that of the cow is rich in this substance. This fact coupled with the more extensive vascular bed may account for the more ready and rapid absorption of the toxic material.

Professor Burrow: I will not join issue with Dr. Amoroso on the relative degrees of intimacy between foetal membranes and uterus in the two species, but agree that the area of contact is greater in the mare and that there is greater opportunity for the absorption of toxins in a short space of time.

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Permeability of the Placental Barrier

What is known of the permeability of the placental barrier to (a) bacteria, (b) drugs, in the deciduate and indeciduate placenta?

Professor O. G. Edholm: The placental barrier appears to be impervious to bacteria, except if a lesion is produced on the maternal side of the barrier. Bacteria may then be able to cross to the foetal side, and affect the embryo. Cases of congenital syphilis in man have frequently been described: if the placenta is examined in such cases, definite lesions are always found.

Drugs, on the other hand, can diffuse through the barrier. The rate of diffusion depends first on the size of the molecule: the larger the molecule the slower the rate of diffusion. But the diffusion rate will also depend on the type of placenta and on the stage that has been reached in pregnancy. The number of layers of tissue between the maternal and foetal circulations varies in different animals. The greater the thickness of the barrier, the slower will be the diffusion rate of drugs. Since the thickness of the barrier diminishes in many animals as pregnancy proceeds it is found that at an early stage, the placenta is relatively impervious to drugs, which will pass comparatively easily across it a late stage of pregnancy.

* See White, E. G. (1938.) *J. Path. Bact.* **47**. 323.

Discussion

Dr. Amoroso: The terms indeciduate and deciduate, though well-entrenched in the literature, are unsatisfactory. They were first employed by Huxley to classify mammals—and do not include the contra-deciduates in which the placenta is retained in whole or in part. Grosse's classification which is based on the proximity of the foetal and maternal blood streams is to be preferred. The four types of placentae which he describes are the epitheliochorial (pig and mare) in which maternal endothelium, maternal connective tissue, uterine epithelium, chorionic epithelium, foetal connective tissue and foetal endothelium separate the two bloods; the syndesmochorial (cow, sheep and goat), in which the maternal epithelium is lacking; the endo-thelicchorial (cat and dog) in which the maternal epithelium and maternal connective tissue are lacking, and, finally, the haemochorial (rabbit and man) in which the chorionic epithelium is directly bathed by maternal blood. It is the proximity of the blood streams which, presumably, determines the permeability of the placental barrier; those placentae in which few layers are interposed (*e.g.*, rabbit and man) allow a more ready transference than those in which there are several layers (*e.g.*, sheep and cow).

During the development of the placenta, it must be remembered that this organ is not static. Its constitution changes during the course of pregnancy; in the rabbit, for example, on the tenth day, the two bloods are separated by three membranes, the foetal endothelium, the foetal connective tissue and the chorionic epithelium; on the 22nd day there are only two of these membranes; and on the 29th day only the foetal endothelium. This placenta is thus haemochorial at the beginning but haemoendothelial at the end. Its permeability varies accordingly.

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Demodectic Mange

Is there any recent information on the life cycle of Demodex canis, *and is there any evidence to support the clinical observation that demodectic mange is hereditary?*

Dr. J. N. Oldham: There is no recent information on the life history of this parasite. The life-cycle of *Demodex canis* is still not well understood and the time required for the completion of the cycle is unknown.

Most workers agree that demodectic mange occurs principally amongst short-coated dogs (Bostons, Dachshunds, smooth-haired terriers and bulldogs) and that symptoms become evident during the first year of life. The uncomplicated squamous type of the disease is the more common and has been shown to occur in about 90 per cent. of animals one worker examined. The mite also occurs in apparently normal dogs without showing skin lesions. About 10 per cent. of clinically normal dogs examined were so affected and the numbers of mites per unit weight of skin in these animals was very much smaller than the number in diseased dogs of comparable age.

A number of workers have shown that the mite occurs in the lymphatic glands of dogs with follicular mange (principally the submaxillary glands, but also in the prescapular and retropharyngeal glands). The parasite occurs in the glands of clinically normal as well as clinically diseased dogs, but apparently never in the absence of a skin lesion. There is evidence to show that the mites are passively transported *via* the blood or lymph stream to the lymphatic glands and the glandular infestation is secondary to that of the skin. There is also some evidence to show that the mites probably neither reproduce in the glands nor are they able to regain the skin from the glands later on.

Several workers have shown that follicular mange is not readily transmitted to healthy dogs. The most recent work indicates inability to transmit the disease in a clinical form to healthy dogs but ability to transmit the mites to uninfected animals by direct skin transfer although not by the oral route. It would appear that the parasite is transferred to the uninfected animal either from the clinically diseased, or the "carrier" animal by direct contact and that in only a small proportion of cases are conditions favourable for the reproduction of the mites.

Difference of opinion still prevails as to whether follicular mange is truly contagious since some people consider the mite as being a harmless saprophyte—or rather saprozoon—in many animals, only becoming pathogenic in a few individuals because two or three puppies in a litter sometimes become affected while the remainder remain healthy. Others consider the disease to be mildly contagious and possibly hereditary (not congenital) inasmuch as it develops in certain strains of dogs, but there is no real (experimental) evidence to support this latter view.

Discussion

Professor Wooldridge: The life history is not known. I have long had the idea at the back of my mind that it is a more complicated life history than for *Sarcoptes*, causing a different type of mange. I suspect that there may be a vector, and the vector lice or fleas. One reason I have had this suspicion is the number of times one has had a case of follicular mange in a particular kennel and the dog is removed and the kennel is left vacant; other dogs are then put into the kennel and within a reasonable time afterwards they develop follicular mange. There is no direct contact and I cannot imagine the parasite lived in the kennel and was sufficiently active to cause a fresh infection which, I think, may have been maintained in the meantime by some vector. Another parasite, *Linguatula*, is remotely related to *Demodex*. It is a degenerate form and the complicated life history is not completely made out so that it does add a little support to the idea that the *Demodex* has a corresponding life history or complicated life history. As to heredity, I have had experience of bitches with no visible skin lesions giving birth to puppies on successive occasions which developed follicular mange within six weeks of birth. I rather fancy in some of those cases although there is no evidence of hereditary infection, the conditions afford an opportunity of the vector transmission or that there is some other means of transmitting the non-clinical affection to the puppies.

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Infestation of Suckling Puppy with Round Worms

How does the young suckling puppy become infected with round worms? Most puppies have these worms and yet the adults appear free from them.

Dr. Oldham: Taking the second part of this question first, it seems that the questioner is in some doubt as to whether adult dogs are really free from worms or not. The position is that *Toxocara canis* is commonly encountered in pups and young dogs, whereas *Toxascaris leonina* occurs in more mature animals. Older dogs may appear to be worm free in that no worms are seen to be passed but faecal examination would probably show that in some instances one or two worms were actually present. Dogs old enough to produce puppies can and in many cases do harbour the common ascarid, *Toxocara canis*. Statistics, at least so far as this country is concerned, are conspicuous by their absence, but two workers in the Far East (Chao, L.[1] and Andrews, M.[2]) have shown that, out of 60 and 590 dogs of all ages they respectively examined there were infections with *Toxocara canis* in 22 per cent. and 35 per cent. Koutz & Rebrassier[3] examined the faeces of 1,486 dogs of all ages from both country and city at the veterinary clinic of Ohio State University and found 16·09 per cent. infected *Toxocara canis*, 5·25 per cent. with *Toxascaris leonina*, and 17·22 per cent. with *Ancylsotoma caninum*.

With reference to the first part of the question, infection of pups before birth has been shown to be possible by Fullebörn[4] and by Shillinger & Cram.[5] This accounts for the appearance of mature or nearly mature worms in very young animals. Since the migration of *Toxocara*—like that of the pig *Ascaris*—is *via* the blood, the larvae undoubtedly reach the placenta through the maternal blood stream and it is generally considered that the larvae are held up in the liver of the foetus until birth occurs when they continue their migration through the lungs and reach the small intestine where they become mature. The evidence of Shillinger & Cram is probably the most conclusive. They used *Toxocara canis*, the eggs of which become infective in about a week. Eggs of this species were fed to a worm-free pregnant bitch eight days before the twelve pups were born. All pups were dead on the day after their birth and microscopic examination of tissues showed *Toxocara* larvae in the liver of six, in the lungs of one, and in the liver and lungs of one of the puppies.

Proper nutrition appears to be a factor in prenatal infection and Wright, W. H., showed that older dogs may be more easily parasitized by *Toxocara canis* if maintained on a diet deficient in vitamin A. It follows that pups born to such dogs are more liable to become prenatally infected than if they were born to a dog whose nutritional level prevented round worms from becoming established in it.

With regard to other dog round worms, Adler, S., & Clark, E. J., found the hookworm, *Ancylostoma caninum*, in six out of 13 dogs, two to 15 days old, and since about five to six weeks are necessary for the development of mature forms of this hookworm after infection, these were undoubtedly cases of prenatal infection.

1 *Chinese Med. J.* (1941.) **59.** 550.
2 *J. Helminthol.* (1937.) **15.** 167.
3 *J. Amer. vet. med. Ass.* (1942.) **100.** 214.
4 *Arch. f. Schiffs- u. Tropenhyg.* (1921.) **25.** 367.
5 *J. Amer. vet. med. Ass.* (1923.) **63.** 200.

Discussion

Dr. Amoroso: Did the examination reveal any lesions in the placenta?

Dr. Oldham: There was no examination for that.

Dr. Amoroso: Structurally the marginal region of the placenta

is simpler than in the labyrinth and in the absence of any demonstrable lesion in the placental labyrinth we may presume that the larvae could only have entered the foetal blood stream through the chorionic processes which project into the lakelet of maternal blood in the green-border.

Dietary Exclusions in Nephritis and Obesity

Can we have an explanation why meat, eggs and fish should be excluded from the diet of dogs suffering from nephritis; starch and salt from obesity cases? The actual reasons for such exclusions have not been apparent to some members.

Professor O. G. Edholm: The exclusion of fish, meat and eggs from the diet of nephritic dogs is an attempt to exclude or reduce the protein content of the animal's food. The products of protein metabolism are excreted by the kidney; so by reducing protein intake the work of the damaged kidney is diminished. However there are several undesirable features of such severe protein restriction. The animal will be malnourished, and will suffer from a lack of the vitamin B complex present in protein foods. At the same time, if albumen is present in the urine, the animal will lose protein, and so may suffer from hypoproteinaemia. For these reasons complete exclusion of protein from the diet in cases of nephritis is not advisable.

In human beings suffering from prolonged starvation, a hypoproteinaemia is found and is responsible for hunger oedema. In treating such cases, as seen at Belsen and elsewhere, preparations of amino acids were used, given either by mouth or intravenously. It is possible that such a method of treatment might be useful in nephritic animals with hypoproteinaemia.

The exclusion of starch or carbohydrates in cases of obesity is based on the fact that carbohydrates can be converted into fat. The specific dynamic action of carbohydrates is small, and so the metabolic rate is not increased greatly by the taking of carbohydrate. If carbohydrate is completely excluded, and the diet consists of fat and protein only, ketosis may be produced. It is more satisfactory to treat cases of obesity by an overall reduction of the food intake, including the fluid intake. It is essential to maintain a proper balance of the various constituents of the food. Since protein has a high specific dynamic action, the proportion of protein in the reduced diet can be varied, so raising the metabolic rate and lowering the weight of the animal. The exclusion of salt I do not follow.

Discussion

Professor Wooldridge: I do not subscribe to the suggestion in the question. In my experience the white of egg combined with milk is almost the ideal diet for nephritis cases. I do not like the use of some products—beef and horse flesh. I exclude the yolk of egg because patients do not do as well as they do with the white of egg alone. Fish, and particularly white fish, is helpful and I would not recommend total exclusion of protein from the diet. I agree with Dr. Edholm that you should rather reduce the total ration and maintain the balance instead of upsetting the ration by omitting any of the proximate principles.

Professor Bosworth: The recommendation to exclude salt from the diet in cases of obesity was based upon the belief that salt favoured the retention of water in the body tissues, a process which it was thought undesirable to allow in obese subjects. I am sure that the recommendation is still given to-day by a good many members of the profession, by teachers as well as practitioners, but whether there is any real reason I cannot say.

Professor Edholm: The point that Professor Bosworth has made about water retention is correct, and in cases of obesity there is considerable water retention. On starving such cases, there may be little loss of weight for 10 to 14 days, then there is a sudden increase in water loss, and the weight diminishes.

Mr. Muir: The temporary exclusion of protein might be advisable in cases of acute nephritis but it is never justified in cases of chronic nephritis where albumen is being lost in the urine.

Connection Between Renal Functions and the Vitamin B Complex

What is the connection between renal functions and the vitamin B complex, since nicotinic acid and riboflavin are being prescribed in large doses of certain forms of nephritis in the human subject?

Mr. W. R. Muir: The kidney is rich in most if not all the known members of the vitamin B complex. This is in conformity with the high metabolic activity of the kidneys and the vital role which vitamins of the B complex play in cell respiration. The high metabolic activity of the kidney will be appreciated when its functions are recalled, namely, the active excretion of the waste products of the body in a solution more concentrated than that of plasma, the regulation of the reaction of the blood and the detoxication of harmful substances in which the kidney plays a role secondary and supplementary to that of the liver. The kidneys also produce a hormone, renin, which can raise the blood pressure, but with that particular function we are not here concerned.

The kidney performs a tremendous amount of work, particularly in concentrating urine, and this is illustrated by a comparison of the oxygen consumption of the kidney with that of other tissues. The following figures are given only for the purpose of this comparison and little attention should be paid to the absolute values. Thus, the kidney of the rat under a given set of experimental conditions was found to consume 25 c.mm. of oxygen per mg. of dry matter per hour compared with a consumption of 15 c.mm. by brain tissue, only 7 c.mm. by liver tissue and 6 c.mm. by resting muscle. If these oxygen consumptions reflect the relative activities of these tissues, then the kidney is very active indeed. Since, as I have said, the vitamin B complex is concerned with cell respiration processes, the richness of the kidney in these vitamins is to be expected. The kidney would also be expected to be sensitive to deficiencies of vitamins of the B complex. It is, but it appears to be much less sensitive than some other tissues. For example, when the diet is deficient, the kidney tends to retain nicotinic acid longer than does the liver. It has been found that, in dogs with experimental "blacktongue," the nicotinic acid content of the kidney is only very slightly reduced, whereas appreciable reduction takes place in the nicotinic acid contents of the liver and skeletal muscles. In apparent agreement with this observation, I have failed to find any report of kidney damage in dogs kept on experimental diets deficient in nicotinic acid or even in naturally occurring cases of "blacktongue" which have not been clearly complicated by other dietary deficiencies or by bacterial infection. Hence, on these grounds, it would seem unlikely that nicotinic acid deficiency is an important factor in the development of nephritis in the dog and one cannot anticipate, therefore, any marked measure of success from nicotinic acid therapy in cases of canine nephritis. I know of no case where it has been of value.

In riboflavin deficiency in dogs there is a certain amount of evidence of tubular and even globular degeneration in the kidneys, but the tissue changes are less striking than those occurring in the liver and in the central nervous system. In the liver extensive fatty degeneration and even necrosis occur. Since riboflavin deficiency does affect the kidneys and since riboflavin plays an essential role in many cell processes, its therapeutic use for nephritis may be justified. But whether riboflavin deficiency is either directly or indirectly responsible for the development of nephritis is rather doubtful in view of the marked changes which occur in the liver and central nervous system in experimental riboflavin deficiency in the dog and which, so far as I know, do not occur in naturally occurring cases of canine nephritis. I do not know the evidence on which is based the recommendation to prescribe large doses of nicotinic acid and riboflavin for certain forms of nephritis in human beings. As far as canine nephritis is concerned, I should say nicotinic acid is likely to have little or no benefit. Riboflavin may have some value but I doubt whether a deficiency of either vitamin is primarily concerned in the development of nephritis in the dog. This question of combined nicotinic acid and riboflavin therapy for nephritis can, however, only be settled by practical tests.

One other member of the vitamin B complex is choline. Choline deficiency has been observed to cause haemorrhagic degeneration of the kidneys in young rats. The curious thing is that spontaneous recovery from this haemorrhagic degeneration and partial restoration of kidney function can take place without correction of the dietary deficiency. I cannot say what part, if any, choline deficiency might play in the development of canine nephritis.

Fresh Grass Giving Rise to Symptoms Resembling Milk Fever

Can the Forum explain why fresh, luscious grass gives rise upon occasion to a train of symptoms resembling milk fever?

Mr. W. R. Muir: Frankly, I do not know. It is known, of course, that grass or lactation tetany in dairy cows is associated with or preceded by hypomagnesaemia, but what is the cause of the low level of blood magnesium and why it may sometimes exist in apparently normal animals is not really known. The condition is definitely and most commonly associated with early lactation and the grazing of young grass although it can occur, but with very much less frequency, in dry cows and bullocks and during stall feeding. Dr. H. H. Green* and the Cambridge workers have dealt with the problem.

Discussion

Professor Burrow: I was always under the impression that there

* Green, H. H. (1939.) *Vet. Rec.* **51.** 1179.
(See also "Minerals in Pasture Deficiencies and Excesses in Relation to Animal Health (1944). Imperial Bureau of Animal Nutrition, Technical Communication No. 15.)

was a connection between hypomagnesaemia in cattle and the manganese content of the grass. Fresh young grass in some pastures has a high manganese content. It is recognized that with the flush of spring grass you do get cases of lactation tetany. In the old days before the hypodermic injection of magnesium salts was instituted we treated such cases by inflation of the udder with filtered air; some cases recovered if treatment was given early and others proceeded to rapid death. In cases of hypomagnesaemia in bullocks, air was injected subcutaneously in the scrotal region.

Mr. Muir: The Cambridge workers have pointed out that lactation tetany is more liable to occur on pastures where the manganese content of the herbage is high. I believe that the injection of manganese salts into rabbits and the feeding of similar salts to cattle and sheep were tried but that these treatments failed to produce any condition closely resembling lactation tetany and, so far as I know, experiments along these lines have been abandoned.

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Inhibition and Promotion of Lactation

What are the methods employed for inhibiting and for promoting lactation, particularly in the bitch? Stilboestrol, for instance, has been credited with exerting both effects.

Dr. E. C. Amoroso: To assure full lactation, three conditions must be fulfilled. There must be:—

(*a*) The requisite mammary development.
(*b*) The initiation of lactation (Lactogenesis).
(*c*) The stimulation of the established lactation (Galactopoiesis).

In respect of mammary development, oestrogen or progesterone alone or together may directly stimulate mammary growth, but optimum mammary development in most species requires the synergistic action of certain pituitary hormones (*e.g.*, the lactogenic hormone, prolactin, the growth hormone and the adrenocorticotrophic hormone). Now, provided there is the requisite mammary development either the adrenocorticotrophic hormone or prolactin in the presence of the other will initiate lactation. For the stimulation of the established lactation prolactin appears to be essential, though as Folley has shown, crude extracts of ox pituitary can increase to a worth while degree the milk yield of cows in the declining phase. The galactopoietic activities of horse pituitary extract though surpassing that of ox pituitary cannot, however, because of its high content of follicle-stimulating hormone, be used in practice. Finally, dried thyroid extracts or the injection of thyroxine not only increases the milk yield but also the milk fat content. Moreover, the combination of certain proteins with iodine (*e.g.*, iodocasein) will also reproduce the galactopoietic effects of thyroxine.

With the elimination of the gestational influences at parturition, suckling initiates and maintains lactation. Moreover when suckling ceases secretion of milk terminates and the mammary glands involute. Ovariectomy and hypophysectomy will prevent the inception of lactation and the latter interference will prevent its continuation if once established.

In answer to the second part of this question we can say that stilboestrol may exert both effects. Large doses will inhibit an established lactation, smaller doses on the other hand tend to stimulate lactation.

Discussion

Professor Ediholm: Thyroid extract is mentioned as having a useful stimulating effect, but it is not so readily available as iodocasein. Iodocasein is relatively cheap and can be used in practice, but one does perhaps want to watch the animal. One will not get a high thyroid effect unless adequate feeding is maintained. I think possibly Mr. Muir will agree that, as far as lactation is concerned, one of the most important things to do is to maintain the level of nutrition. With stilboestrol, Parkes and others have stimulated lactation by implanting pellets of this synthetic oestrogen deeply under the skin.

Mr. Muir: It is important to realize that the action of stilboestrol in inducing mammary development and lactation is a slow one since time is needed for the development of mammary tissue. I emphasize this fact because someone once asked me whether stilboestrol could be used to induce lactation in a dry bitch so that she could suckle a litter of puppies whose mother had died at parturition. My questioner had not realized that lactation in response to stilboestrol treatment could only follow a period of mammary duct and alveolar development. Inhibition (or stimulation) of an existing lactation is much more rapid. Iodocasein, it is true, will stimulate lactation in respect of both yield and composition of milk, but as some of you know, the Ministry of Food has issued an order prohibiting the inclusion of iodocasein in manufactured feeding-stuffs. I believe that this order was made on account of the results of feeding experiments with iodocasein to dairy cows on ordinary commercial farms. I believe that adverse effects were observed on the health, body weights and eventually milk yields of some cows. This is not surprising, perhaps, since, of necessity, the nutrition of dairy cows cannot always be maintained at a high level on, at least, some commercial farms. When thyroid active substances are administered and the whole level of metabolism thereby raised, nutrition must be maintained on a high level otherwise the effects will be disastrous. In our present state of knowledge, at least, I have little faith in the hormonal stimulation of lactation on commercial farms. The real opportunities for improvement in lactation lie in the nutrition during pregnancy. If this is fully adequate, then in most animals at least the hormonal system will function normally and, at parturition, normal lactation will commence. In particular, the level of milk production in the early stages of lactation is highly dependent upon the level of nutrition and the reserves accumulated during pregnancy. So also to some extent is the persistence or duration of lactation. In animals, not specially bred for lactation, the effects of nutrition during pregnancy upon the subsequent lactation and capacity to nourish the offspring adequately appear to be particularly marked quite apart from and independently of the level of nutrition after parturition.

Dr. Amoroso: To give stilboestrol early in pregnancy in the hope that it might stimulate mammary development is a practice which must be deprecated since it may cause degeneration of the eggs during the first week after fertilization.

Professor Bosworth: It is said that the fracture of the pelvic bone in cattle subjected to stilboestrol treatment is not due to impairment of the structure of the bones, but is merely an accident arising from the bulling. I think the question is one which certainly demands further investigation from the pathological point of view. In mice there are definite changes in the bone.

Mr. Muir: I support that. There is evidence, particularly in the case of birds, that bones in certain regions are decalcified to some extent by oestrogens. This question of the effect of stilboestrol on the structure and mineral content of the pelvic and other bones certainly requires investigation.

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Thyroid Gland Relationship with Female Sex Hormone

What, if any, is the interrelationship between the thyroid gland in the production of the female sex hormone? Could the relationship be responsible for the typical hypothyroid symptoms of alopecia, skin irritation and lethargy which occur in some animals following each heat period?

Dr. E. C. Amoroso: There is no evidence of any direct effect on the production of the female sex hormones. The activity of the thyroid gland is cyclic and this cycle is closely connected with the genital cycle. This seasonal variation is primarily concerned with the regulation of the animal's general activity and metabolism. Since during the period of low activity, the thyroid can be stimulated by anterior pituitary, even to a greater extent than that which the gland exhibits at high activity and since castration and the administration of the sex hormones has no such effect, it appears that the anterior lobe constitutes a factor of significance in the manifestation of the thyroid rhythm. By means of experimental hyperthyroidism, however, pseudopregnancy has been produced in the albino rat; abortion and foetal resorption usually occurred when the animals were pregnant. Two explanations may be advanced. The desiccated thyroid could stimulate the anterior pituitary to produce hormones which bring about the formation and persistence of large functional corpora lutea or, on the other hand, the heightened metabolism resulting from thyroid administration may lower the oestrin concentration to such a degree that the function of the corpora lutea may be expressed.

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Detoxication of Nembutal

Why do some young, healthy dogs take up to 24 hours to come round completely after intravenous nembutal anaesthesia, although weighed accurately and dosed according to weight or even slightly underdosed? On the other hand, some dogs known to have, for example, nephritis are conscious and co-ordinate in the usual eight to twelve hours.

Professor G. H. Wooldridge: The question is an awkward one to answer. Judging by my own experience, I should say it is a matter of idiosyncrasy and nothing else. As far as I know, there are no means of determining how or why that peculiarity of the individual exists. The same thing takes place in the application and administration of other drugs. One individual will react differently from another. For example, morphia when administered to some animals acts as an excitant instead of a narcotic. I should say, therefore, that the varied reaction is a matter of idiosyncrasy.

Discussion

Mr. Muir: Mr. G. Knight, of the Beaumont Animals' Hospital, has suggested to me that the rate of injection plays a responsible

part in the varied reaction of different dogs to intravenously administered nembutal. Mr. Knight says that the slower the rate of injection the less is the effect of a particular dose of nembutal. When the patient shows activity and excitement, there is a tendency, unconscious or otherwise, to give the injection at a faster rate than when the patient is quiet and passive. Hence, although the dose of nembutal may be standardized, it is not likely in practice to be injected at a constant rate to all patients and so one would expect reactions to vary quite apart from differences due to idiosyncrasy.

So far as I can recall the published results of experiments on the excretion of barbiturates, the excretion of these drugs in the urine is delayed in nephritic patients and, consequently, such patients tend to recover more slowly from anaesthesia. This observation is the absolute reverse of the experience indicated by the question. Probably the type of nephritis is important. A glomerular nephritis might favour retention whereas tubular nephritis might not markedly affect excretion.

Professor Wooldridge: In cases of chronic nephritis it is unsafe to use nembutal.

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Fatal Sequelae to Stilboestrol Therapy

Why is it that so many fatalities occur as a sequel of stilboestrol therapy, particularly in bitches suffering from chronic metritis?

The Chairman: Professor Wooldridge left early, but he asked me to say that he was not in a position to accept or repudiate the implication stated in the question. He has never used stilboestrol for these cases of chronic metritis. He finds that he gets good results from the use of pituitrin.

Discussion

Dr. Amoroso: If true, it might be explained by assuming that stilboestrol, which can be detoxicated by the liver under normal conditions, may accumulate, because of the systemic effect of chronic metritis, in quantities sufficient to exert a deleterious action.

Mr. Muir: Stilboestrol has been recommended for the treatment of chronic metritis on theoretical grounds which, at first sight, would appear to be reasonable; nevertheless, I believe that those same grounds could be challenged. However, the question is why are cases of chronic metritis so likely to be fatal following stilboestrol therapy. I would suggest that the condition of chronic metritis so reduces the powers of resistance of the animal body that it is unable to deal with stilboestrol which is really a toxic as well as a physiologically active substance. In consequence, I would expect the liver to suffer fatal injury. The point is that the resistance of the body depends very largely upon the detoxicating mechanisms of the liver and kidney. For the processes of detoxication, the body uses amino acids obtained mainly from proteins stored in the liver; glycogen, vitamins of the B complex, vitamin C and possibly even vitamin A may be indirectly concerned. Some of these substances (*e.g.*, amino acids and glucose) may be combined directly with toxic substances in order to render them harmless while the vitamins, especially those of the B complex, for example, riboflavin, are mainly concerned in oxidation processes associated with detoxication. Destruction by oxidation, I believe, is one important means of removing stilboestrol from the body. The resistance of the liver to any toxic substance largely depends therefore on its reserves of proteins, glycogen and vitamins. The existence of a chronic condition like metritis makes a continuous demand on the materials of detoxication and tissue replacement and may even exhaust the body's reserves. This depleting effect of a chronic disease can be counteracted only by an ample supply of good quality proteins and vitamins in the diet. In this connection, it is important to remember that the liver's store of vitamins of the B complex cannot be greatly increased without the simultaneous deposition of protein. Since detoxication takes place mainly in the liver and kidneys, these organs are likely to suffer injury when their protein, glycogen, and vitamin reserves are low. I would expect chronic metritis to reduce these reserves to a low level. The liver would therefore be in a poor condition to withstand the toxic effects of stilboestrol when this substance is administered in the fairly large doses which I believe are used for the treatment of chronic metritis. If this argument is true, then it is not surprising that deaths frequently occur.

Professor Edholm: Can anyone tell us about the mode of death?

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General Discussion

Mr. G. E. Joss: With regard to the question about fatalities due to stilboestrol, some time ago I always gave stilboestrol in cases of pyometra. In all these cases, severe abdominal pain was reported about 24 to 48 hours after administration, whether by mouth or by injection. Most cases collapsed and hysterectomy was performed immediately. In all these cases where stilboestrol had been given, I noticed that haemorrhages, sometimes very extensive, were present between the serous and muscular layers of the uterus. On one occasion a haemorrhage was present on the small intestine. Since these findings I have never used stilboestrol for pyometra. Have these haemorrhages anything to do with the fatalities recorded?

Miss J. O. Joshua: In about 50 per cent. of such cases comparatively sudden death occurs when stilboestrol is given, usually about 48 hours after the commencement of the treatment. I presume that as the uterus is brought into a condition resembling oestrus there is considerable hyperaemia, possibly with increased permeability of the uterine wall, and that in cases where there is bacterial infection of the discharge an actual bacteraemia could result, death being virtually from septicaemia. I have also had a suspicion in some cases that stilboestrol has had a deleterious effect on cardiac action; of this, however, I have no definite evidence.

Dr. T. Hare: I think the question put to Dr. Amoroso about the thyroid was incorrect. I would suggest that the signs indicated in the question are those not of hypothyroidism but of chronic toxaemia. I would suggest that in general practice hypothyroidism is rare in adult female dogs. On the other hand, the syndrome mentioned in the question is very common and I think it will be found to be the result of toxaemia, consequently thyroid medication does not arise.

I suggest that Miss Joshua's observation of the death of dogs following stilboestrol medication is due directly to failure of the liver which has degenerated as a consequence of toxic absorption over a long period. The organ which takes the strain in a long-continued toxaemia, even before the appearance of the pyometra, is the liver.

Professor Burrow made an interesting observation about the absorption of the *toxic* products of tissue breakdown; but is there proof that natural tissue breakdown within the living body is toxic? Also, I disagree with his suggestion that the cow's resistance to streptococci is greater than that of the horse. I would say both animals are equally susceptible but the individual's susceptibility varies according to many factors, such as age, dose of infection, and how long that animal has been previously exposed to the infection. It is possible that a cow has been suffering from streptococcal infection for the greater part of its life so that by the time of parturition a certain degree of natural immunity has been attained. In strangles, the young horse and mare go down with acute streptococcal infection whereas the old brood mare having had the infection over a long period is relatively a much milder clinical case.

I did not understand Dr. Oldham's remark about demodectic mange. He said 90 per cent. of dogs are infected; but it does not exist to anything like that degree in nature. I should say demodectic mange is relatively infrequent. I think it would be unfortunate if it went out that amongst dogs in this country the incidence of demodectic mange is high. In a particular untreated kennel, a high incidence may occur, but in the general canine population the incidence is not high.

Mr. J. N. Ritchie: I understand from Dr. Amoroso that some animals do not shed the placenta and that at least one domestic animal retains it in part. Could he tell us which are these animals?

Dr. Amoroso: In the case of the sheep and the goat, there is a much closer association between the foetal and maternal blood stream than in the horse and pig. In the development of the placenta of the sheep, the uterine epithelium is destroyed. No such destruction occurs during the development of the placenta in the mare and sow. In the sheep, the uterine epithelium of the crypts is replaced by foetal tissue which remains *in situ* at the time of parturition. In the case of animals (contradeciduate types, *e.g.*, tree-shrew, marsupials) where the whole of the cleansing is retained, the placenta is reabsorbed *in situ*. The histological features which characterize the placenta of the cow and mare are not found in rodents (mouse, rat, guinea-pig, rabbit), in which the embryos do not depend to the same extent on the secretion of uterine milk for their nutrition during the early stages of development.

Dr. Hare: The amount of tissue destroyed and absorbed by the horse is enormous and works out at a considerable figure after a normal eight- to ten-hour day.

Mr. Muir: The autolytic breakdown of formed tissues in the body or uterus is not, I believe, known to result in the formation of toxic substances and I think this is unlikely to occur. On the other hand, when bacterial infection exists in the autolyzing tissue or at any other site in the body, substances of varying degrees of toxicity are produced as a result of the metabolic activities of the bacteria.

Dr. Amoroso: With regard to the permeability of the placenta, we know that, in the case of the cow and the sheep, very little vitamin A crosses the placenta and that much of the vitamin A of the newly-born animal is obtained from the colostrum in the first few days of life. One day's feed of colostrum from the cow

CLINICAL COMMUNICATION

Extra-uterine Pregnancy

P. L. BROWN, B.SC., M.R.C.V.S.
CHIPPENHAM, WILTS.

SUBJECT: Shorthorn heifer, aged 2½ years, 7½ months in calf.

HISTORY, SYMPTOMS AND DIAGNOSIS:

5.9.46.—The heifer was first noticed to be unwell by the owner. She was hunched up; feeding and cudding were suspended and the bowels costive. She was given one pint of linseed oil.

6.9.46.—No improvement, and advice was sought. Examination revealed the following:—

Temperature 103·5°; pulse fast and soft, 110 per minute; respirations slightly accelerated; slight tympany, no detectable rumen movements; faeces normal to loose; breath smelling strongly of proprietary medicine. There was diffuse pain all over the epigastrium; a painful grunt was elicited by pinching the back and by pressure over the xiphoid region. The animal walked reluctantly with back level.

A tentative diagnosis of indigestion was made. Sodium bicarbonate 8 oz. and ammon. carb. ½ oz. *b.i.d.* was prescribed.

7.9.46.—A marked improvement with the resumption of cudding and feeding, and an absence of tympany, was reported by the owner.

8.9.46.—Condition had reverted to that seen on the first visit. For this the owner blamed himself as he had omitted the sodium bicarbonate when the heifer had seemed so much better. Treatment was continued on the same lines with a warning that the trouble might be due to the presence of a foreign body and that an operation might be necessary.

10.9.46.—The animal was again seen and this time a small quantity of discharge was noticed on the tail and she was examined *per vaginam*. The os was partly dilated and the allanto-chorion was bulging through it. She was left for 24 hours when it was thought that the act of abortion would have been completed.

11.9.46.—The animal was very blown and distressed. She was discharging a foul-smelling bubbly fluid. On examination, portions of putrid foetal membranes were withdrawn but there was no sign of a calf. Beyond the cervix and on the floor of the left uterine horn was a rent with firm edges and the foetal membranes could be felt disappearing through this rent.

The animal was shot and a *post-mortem* examination carried out.

Post-Mortem FINDINGS: The rent in the floor of the uterus was about five inches long with thickened fibrous edges. There was a diffuse acute peritonitis. The foetus was about eight months and undergoing decomposition. The foetal membranes had formed a placental union with the peritoneal surfaces of the rumen, adjacent intestines, and with the mesentery. This adventitious placenta was in well-defined cotyledonary areas.

DISCUSSION AND CONCLUSIONS

Rupture of the uterus had apparently taken place some considerable time before symptoms were shown, the foetus carrying with it its intact foetal membranes. It is possible that part of the foetal placenta became detached from the maternal cotyledons, and that these areas had provided the stimulus for the formation of similar cotyledonary areas on the peritoneal surfaces of the rumen, intestines and mesentery. No naked maternal cotyledons were found in the uterus, but this may have been due to the formation of addition foetal cotyledons opposite the denuded maternal counterparts after rupture had taken place. Alternatively, only a portion of the allanto-chorion devoid of areas of villous growth may have passed through the rupture and in time formed cotyledonary union with the peritoneal surface of adjacent viscera. This seems less likely as placentation was not diffuse.

It has been regarded as fact that villous formation of the allanto-chorion opposite predetermined cotyledonary areas in the uterus was a growth reaction incited by the uterine endometrium at these points; but in this case an extra-uterine adventitious cotyledonary placenta was formed, presumably at the stimulus of the allanto-chorion.

Infection had obviously gained entry when the cervical seal broke down and had resulted in an acute peritonitis.

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has been estimated to furnish as much vitamin A as the milk supply for approximately 50 days. Antibodies, which are proteins, cross the placental barrier in haemochorial types, but there is no evidence to show that toxins themselves get across.

Professor BOSWORTH: Antibodies certainly do not cross the placental barrier in the sheep. In the case of lamb dysentery, it is well known that lambs of immune mothers obtain their antitoxin from the colostrum.

ABSTRACTS

[The Antidotal Treatment of Arsenical Poisoning. PIERCY, S. E. (1946.) *J. comp. Path.* **56**. 237-245.]

Arsenical poisoning of livestock is of considerable importance in Africa because of the losses occurring from the widespread use of arsenical dips for controlling tick infestation and of sodium arsenite baits for locusts. As test animals the author used healthy young steers given sodium arsenite as a drench in a dosage equivalent to 0·4 g. As_2O_3 per 100 lb. body weight and this usually caused death after about three days. Recently precipitated ferric hydroxide, the classical antidote, was effective in preventing death only when given within five minutes of the lethal dose of As. Sodium thiosulphate in doses of 12 to 24 g. by mouth, together with 2·0 to 5·6 g. intravenously, repeated two or three times during the succeeding 36 hours, was effective in preventing death when administered up to twelve hours after the sodium arsenite. Since under field conditions it is likely that the amount of As ingested will often be less than in these experiments, sodium thiosulphate may possibly be effective even when its administration is delayed longer than twelve hours. Neither antidote is likely to be effective when several times the minimum lethal dose has been ingested or when marked clinical symptoms already have appeared.

E. G. W.

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[An Apparently New Transmissible Disease of Cattle. OLAFSON, P., MACCALLUM, A. D., & FOX, H. F. (1946.) *Cornell Vet.* **36**. 205-213.]

The condition described in this paper, which was presented at a meeting of the New York State Veterinary Medical Association in July, 1946, is an acute, contagious and transmissible disease of cattle, characterized by leukopenia, high temperature, salivation, nasal discharge, diarrhoea, depression, anorexia, and dehydration. The lesions included necrosis and ulceration of the mucous membranes of the lips, cheeks, tongue, pharynx, and oesophagus. The effects of the disease in a herd were loss of weight, drop in milk yield, abortion, and a mortality of 4 to 8 per cent. The first cases were encountered in March, 1946, and six herds were affected, the incidence varying from 33 to 88 per cent.

The disease was transmitted to eight- to 14-month-old heifers by drenching with faecal material, by subcutaneous injection of blood from affected animals at the height of the febrile phase, and by injection of spleen emulsion from animals destroyed during the febrile phase. Material which was infective for calves had no effect on mice, rabbits, guinea-pigs, sheep or chick embryos. Experimentally infected cattle showed a rise in temperature after seven to nine days which lasted for three to six days. Salivation, nasal discharge and scouring occurred after about ten days and the animals very rapidly lost flesh and became dehydrated. Some three days after scouring occurred ulcers appeared in the mouth: in animals which recovered the ulcers healed rapidly. Sometimes the most vigorous experimental animals died within two or three days of the onset of scouring. Diarrhoea and leukopenia did not occur in all the animals.

Symptomatic treatment, consisting of transfusions of blood or saline, were used: penicillin did not appear to be of value and sulphonamide drugs were not used because of the leukopenia.

Conditions which are considered in differential diagnosis of the disease are winter dysentery (less duration and not so severe), shipping fever (coughing and pulmonary lesions), and acute poisoning due to caustic agents (immediate mouth and oesophageal erosions).

E. G. W.

WEEKLY WISDOM

Beauty is truthfully an expression of hope and that is why it is so enthralling. . . . Though I cannot name the ideal good, it seems to me that it will be in some way associated with the ideal beauty of nature.—RICHARD JEFFERIES.

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Last year's Congress visitants to the A.R.C. Compton Estate will be interested to learn that Mr. Eric MacMillan, the farms manager, whose fund of knowledge added so much to the interest in its agricultural lay-out and management, is leaving to take a post at Norwich. He has been at Compton for nearly 30 years, and at one time was farm manager for the late Mr. Alfred Barclay.

IN PARLIAMENT

Report of the Food and Agricultural Organization of the United Nations

On the motion for the adjournment of the House of Commons on February 6th, Mr. Wilson, Parliamentary Secretary, Ministry of Works, opened a debate on the report of the Food and Agricultural Organization of the United Nations. He said that he intended to deal with the report of the F.A.O.'s preparatory commission on world food supplies. One of the most important steps towards securing increased production of food was to give farmers a real assurance that their increased output would not lead to the terrible collapse in prices which brought ruin and distress to agricultural communities all over the world in the 1920s and 1930s. Even to-day, while the need was still for more food, the one thing dominating the minds of farmers in the Middle West, Canada and Australia was the fear of future surpluses and an inability to find a market for their increased output. With the greatly enlarged capacity to produce food—in the United States it was 40 per cent. above pre-war, even with less manpower, and there was a similar growth in capacity in Canada and in this country—Sir John Boyd Orr felt that only by expanding the mass consumption of the ordinary peoples of the world could the problem be solved; that we needed a world food board to hold stocks of the more important foods, to create a famine reserve, and to provide funds for financing the disposal of surplus agricultural products.

In many ways the danger to-day was even greater than in the 1930s. Potential world output when the war-devastated areas got back into production would be much greater than before the war, and steps taken by Governments in food producing countries to provide guaranteed prices and assured markets would create extremely serious problems which could only be solved by international planning. There was hardly a Government which had not introduced measures of this kind. The Government felt strongly that any international tendency to supervise international commodity arrangements should go wider than the F.A.O. and should be associated with any international organization for the promotion of full employment policies and the expansion of world trade. The Government had no intention of going into agreements on the basis of excessive prices from the beginning of the agreements, and there was certainly no intention of maintaining indefinitely some of the prices ruling some markets to-day.

The Government attached great importance to the proposal for linking potential surpluses for the needs of the under-fed peoples. The greatest efforts must come from the peoples and Governments of the undeveloped countries, and in the case of dependent territories from the Governments of the responsible countries.

In order to bring the whole field of world agricultural and nutritional products within the scope of an agreed international plan it had been proposed that each nation should submit to the annual conference of the F.A.O. its programmes for all important food and agricultural products. It would then be possible to marry up these programmes and see that they were properly linked together.

The report represented the unanimous agreement of the 16 nations on the Commission and it was free from any individual reservations or disagreements. Yet it contained sufficient concrete proposals for providing a new deal in the world food and agricultural situation and a real hope that when the present crisis was over we should go forward to something very different from what we knew before the war. The report was now in the hands of the various member Governments of the F.A.O. for acceptance and action. The Government fully accepted it as a basis for world food planning in the future.

It had been said that these proposals drew the teeth from the Boyd Orr scheme. But they had taken that great conception and produced from it a practical scheme which they believed would work. So far from taking the teeth out they had put teeth in—strong teeth so fitted that they were not likely to bite off more than they could chew.

Mr. Law said that the main purpose of having the debate at that time was for the Government to give a decent, quiet, and unpretentious burial to the proposals of Sir John Boyd Orr.

Mr. Wilson said he had seen Sir John Boyd Orr recently and he had expressed himself as highly delighted with the report of the Commission.

Mr. Law said there was no doubt that this plan did end the executive World Food Board that Sir John Boyd Orr had proposed at Copenhagen. In the view of the Opposition the plans for buffer stocks were sound. It was essential to iron out the violent fluctuations in the prices of primary commodities.

The form of buffer stocks proposed at Washington was extremely dangerous in that it contained the vicious principle of differential prices. As it stood the proposal was fraught with the greatest danger to the people of this country, and would tend, inevitably, to reduce rations in this country not only in an emergency but over a long term. The people of this country deserved something better. The Government should look at the proposal again—remembering that their first duty in the matter was to consumers in this country, who were having an extremely difficult and harsh time.

Mr. W. Roberts said that if out of the present famine conditions production was increased until there was a glut we might find ourselves back in the 1929 situation much faster this time than after the 1914-18 war. He thought there was danger of that happening. It was vital there should be more international trade and that Governments should practise an increasingly expansionist rather than a restrictive policy. The idea of a planned interchange of industrial and agricultural products between countries need not be only a dream. He was not convinced that the organization here set up could make it practical.

Farmers and a Guarantee

Mr. Eccles said he thought it was likely that his hon. friends would have divided against the Agriculture Bill if they had known that the Government were going to put their name to a report which forecast such enormous surpluses of crops of the type we grew here. They would have insisted on a guarantee as to the proportion of the home market which the British farmer was to fill.

If we were going to comply with the White Paper on the American loan and divest ourselves of all possible quota and tariff weapons on our manufactured products while the F.A.O. report was to be applied to everything we bought then multilateral trade was not possible. That was not multilateral trading at all; it was the worst of both worlds. The F.A.O. report was a sell-out to the United States Government. It was not that he did not want to raise the nutritional standards throughout the world, but to ask Britain to pay far more than her share in her poverty-stricken condition and to do it under international treatment was simply not right. Mr. Strachey was to be congratulated on earning a good post in the United States administration. This system provided the American farm *bloc* with an outlet they otherwise would not have had. He disliked more than he could say the Government putting their name to a paper knowing the British delegation could not have put British interest forward with that tenacity and understanding expected of any Government.

Mr. Reid said that if recent developments meant that bulk buying was going to be less effective than it was, then it was the duty of the Minister to revert to other and well-tried methods of buying. If by reason of ideological considerations he refrained from so doing and thereby lost food for the people he and the Government were incurring a very heavy responsibility.

He doubted whether international agreements of this complexity could be made in the absence of Russia and those countries which took their cue from Russia. The success of all these and corresponding agreements depended fundamentally on solving the question of dollars and unless we could solve that all the report was as near as might be waste paper. For that reason he deplored the light-hearted manner in which the Government were treating our dollar resources. The Minister should redouble his efforts to reduce our dollar expenditure to reasonable proportions.

Mr. Strachey's Reply

Mr. Strachey, Minister of Food, said that in spite of all the very great difficulties food prices only stood at 22 per cent. above the pre-war level. If a wider range of foodstuffs were taken in than were included in the index figure they were certainly not above 33⅓ per cent. over pre-war. After the 1914-18 war, with other methods, at the peak point at the end of 1921 food had almost trebled. That was an indication of the degree to which the Government had been able to hold prices down. He did not say they would be able to carry that through without some increase. If inflationary pressure in the world went on indefinitely they might have to give ground before it

Whatever else the differential price arrangements for needy countries could affect, they could not affect the quantity of foodstuffs brought into this country. They might affect the price, but in conditions of extreme surplus it was a most remote contingency that we would be unable to pay. Such proposals would be scrutinized most carefully by the Government and only if they were hedged round by most careful provisions would they subscribe to them.

Subsidy provisions for countries with special nutritional problems applied equally to this country. It might be argued that if such a scheme were not in force prices would go lower. That was perfectly true, but it was possible to import wheat too cheaply and drive too hard a bargain with primary producers. In ruining them one ruined one's best customers.

Another important consideration was what would happen if we

refused to have anything to do with the arrangements, after most careful safeguards. Schemes of exclusive restriction would at once be adopted by the great producers who, instead of sending wheat at a specially cheap price to needy consumers, would plough surplus crops into the ground.

Although this country as a whole was consuming about 7 per cent. less food than before the war, there had been an immense redistribution. The upper third of the community—the *rentiers*—had found their diet reduced by much more than 7 per cent.; the middle third consumed about the same; and the poorest third was consuming far more than before the war. Although eggs and bacon were in low supply, other important foodstuffs were being consumed in appreciably greater quantities. The quantity of milk consumed was 147 per cent. higher than before the war. The amount of cheese consumed was about the same as before the war.

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Veterinary Services (Meat Inspection), (Slaughterhouse Conditions)

Mr. G. Williams asked the Minister of Agriculture whether, with a view to the better control of disease among livestock and an improved standard of meat, he will make arrangements in the near future to ensure a more effective co-ordination of veterinary services concerned with meat inspection and animal health, respectively.

Mr. T. Williams: Meat inspection is carried out by officers of local authorities, with whom liaison is maintained by the Ministry of Food through its technical officers. All practical steps are taken to maintain effective co-ordination between these officers and the veterinary officers of my Department.

Mr. G. Williams: Is the Minister aware that there is a considerable shortage of veterinary surgeons, and is he taking any steps to increase their number?

Mr. T. Williams: Yes, Sir.

Mr. Beechman: Will the Minister, in association with the Minister of Food, cause an annual survey to take place of slaughterhouse conditions, so that we may have a policy on these matters for the whole of the country?

Mr. Williams: There was a policy before the war, which had to be suspended during the war, when some 16,000 slaughterhouses were reduced to 600. I am hopeful that it will be possible to make a further review.

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Central Abattoir System

Sir P. Macdonald asked the Minister of Agriculture whether, in view of the present enquiry into the marketing of agricultural products, he will consider creating a central abattoir system under veterinary control which would assist in eradicating disease from farm livestock.

Mr. T. Williams: The veterinary aspects of an abattoir system are undoubtedly important and, though they may be of secondary importance to the economic aspects, they will not be overlooked when decisions on future slaughterhouse policy are taken by the Government.

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Sir R. Glyn asked the Minister of Food whether, in order to reduce congestion and consequent cruelty to animals sent for slaughter, he will now consider the advantage of allowing 50 per cent. of the licensed slaughterhouses in each local authority area to be licensed that were licensed in 1938, as such action will improve the quality of the meat and reduce the demands on transport.

Dr. Summerskill: We are prepared to open more slaughterhouses where suitable premises are available and where this will contribute to the efficiency of the meat control scheme, but unfortunately suitable premises are not available in the localities where most of the recent criticisms have arisen.

Sir R. Glyn: Would the hon. Lady consider appointing a specialist committee to go into this matter in view of the fact that when slaughterhouses were closed as a war measure, there was no indication that it would become permanent policy, and will the matter be looked into from the point of view of efficiency in every local district?

Dr. Summerskill: We have our experts enquiring into the matter and I am giving it my personal attention. It will of course become part of our long-term meat policy which we are considering at the moment, and when this has been decided we shall make an announcement concerning slaughterhouses.

Mr. Quintin-Hogg: Will the hon. Lady give the House an assurance that in considering this matter the Government will look at it not merely from the point of view of the efficiency of their meat scheme but from that of humanity to animals?

Mr. Bossom: Will the hon. Lady review all those slaughterhouses which were closed during the war to see if they could be used at present now that the situation is becoming very serious?

Dr. Summerskill: We have looked at every one, but, I would remind hon. Gentlemen that many of these slaughterhouses were in the backyards of butchers' shops and that the entrance for the animals was through the shop. We have no intention of opening those again. We have re-opened all those which are suitable.

Mr. Vane asked the Minister of Food whether he will ensure that the new slaughterhouses required are built in country districts where the cattle are finished, rather than in urban areas.

Dr. Summerskill: The question of siting of slaughterhouses is one which will have to be considered along with other relevant factors as regards future slaughterhouse policy, and I regret that I am unable at this stage to give the assurance asked for.

Sir R. Glyn asked the Minister of Food what was the total number of licensed slaughterhouses in 1938; what was the number in January, 1947; and what were the respective totals of animals sent for slaughter in those periods.

Mr. Strachey: Before the war, private slaughterhouses were licensed by local authorities and I regret that I have no precise information as to their number. It has been estimated that the number of slaughterhouses in use in 1938 was about 16,000. The slaughterhouses now in use number 606. Animals slaughtered for human consumption in 1938 numbered 1,924,000 cattle, 837,200 calves, 9,963,200 sheep and 2,589,600 pigs. I regret that the numbers of animals slaughtered in January, 1947, are not yet available, but I will let the hon. Member have the figures as soon as possible.

At a later session Sir T. Moore asked the Minister of Health whether he is aware that Memorandum No. 62, Food, which lays down conditions for the condemnation of carcasses at slaughterhouses is unsatisfactory in effect because its provisions do not have the force of law; and whether he will consider introducing for England and Wales regulations to deal with this matter on the lines of the Public Health, Meat Regulations, in force in Scotland.

Mr. Bevan: The control exercised by the Ministry of Food ensures in practice that meat for sale for human consumption is made available for inspection at the time of slaughter, and under these conditions I see no advantage in the course suggested.

Sir T. Moore: Surely the Minister is aware that Scottish methods are much more satisfactory than those adopted in England and Wales, and will he not adopt the Scottish regulation in this matter and so improve the health of the community?

Mr. Bevan: I hope the hon. and gallant Member will not incite bad feeling between the three countries in this matter.

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Tuberculosis (Bovine Infection)

Mr. Granville Sharp asked the Minister of Health what approximate percentage of human tuberculosis is attributable to bovine infection; how many deaths occurred from this cause during each of the last five years for which records are available; what powers he has to protect the public from this source of infection; and when he proposes using them to the full.

Mr. Bevan: It is estimated that bovine infection causes less than 2 per cent. of pulmonary cases, 30 per cent. of non-pulmonary cases, and 6 per cent. of all tuberculosis deaths in England and Wales. No powers at present exist to prevent the sale of tuberculous milk until the infected cow is discovered, but the question is under review by the Departments now considering revised Milk and Dairies Regulations.

Mr. Sharp: Can the Minister say how long this matter has been under review and when a decision may be expected?

Mr. Bevan: Very shortly, I hope. It has been under consideration for some time, but it is a complicated matter.

Mr. Somerville Hastings: Is not the right hon. Gentleman aware that most of these deaths could be prevented by the pasteurization of milk?

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Cruelty to Animals Act (Inspectorate)

Sir G. Fox asked the Secretary of State for the Home Department, whether he will provide information as to the nature of the training in scientific methods and animal husbandry of those inspectors appointed by him under the Cruelty to Animals Act, 1876.

Mr. Ede: If the words in the Question refer to special courses of instruction in the management of farm animals, which are arranged for students seeking degrees or diplomas in agriculture, dairying or veterinary medicine, the answer is that the medical men appointed to assist me in the administration of the Act of 1876 are not required to have taken such courses.

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B.M.A. Negotiations

Lieut.-Colonel Sir Thomas Moore asked the Minister of Health

what progress he is making in his negotiations with the B.M.A. in regard to the administration of the National Health Service Act.

Mr. Bevan: I have invited the negotiating committee to meet me, and I am awaiting their reply.

Sir T. Moore: Is the right hon. Gentleman satisfied that the leaders of the three professional organizations represent the bulk of the British Medical Association members?

Mr. Bevan: I think that it would be impudent as well as imprudent on my part to express an opinion on the heads of the medical profession.

Sir T. Moore: That is a change of heart.

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Foot-and-Mouth Disease (Compensation)

Mr. Digby asked the Minister of Agriculture whether he will pay to farmers, whose herds have been slaughtered owing to foot-and-mouth disease, compensation to cover consequential losses incurred by them, before their farms can be re-stocked.

Mr. T. Williams: No, Sir. The Diseases of Animals Act provide that compensation payable on slaughter shall, in the case of an animal affected with foot-and-mouth disease, be its value immediately before it became so affected and, in all other cases, be its value immediately before slaughter.

Mr. Digby: Is the Minister aware that the farmers affected still have many current expenses to meet, such as wages bills, that they cease to receive the milk on which they largely rely to meet their expenses, and that as a result considerable hardship is caused?

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Pedigree Livestock (Veterinary Tests)

Mr. O. Poole asked the Minister of Agriculture if he will take steps to ensure that veterinary tests carried out on pedigree livestock in the United Kingdom and Canada conform to the same standards.

Mr. T. Williams: There are practical difficulties in this proposal, but it is hoped to discuss the matter with the Canadian authorities in the near future.

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Poultry Industry

Lord Willoughby de Eresby asked the Minister of Agriculture whether, in view of the importance of the poultry industry to this country and the Government's policy to further its expansion in due course, he will ensure that the industry is no longer counted or referred to as a subsidiary agricultural occupation by his Department.

Mr. T. Williams: I fully recognize the national importance of the poultry industry and can assure the noble Lord that if there are occasions on which it is described as "subsidiary"—and I cannot guarantee there will not be—the word is not used in any derogatory sense, but merely as a reflection of the fact that a large proportion of our egg production has been, and probably always will be, ancillary to the main farming enterprise on the holdings from which the eggs are produced.

Lord Willoughby de Eresby: In view of the fact that the poultry industry is a matter of major concern to a great number of people in this country—consumers as well as producers—does not the Minister think that the moment has arrived to raise the official status of the industry?

Mr. Williams: It is not my desire or the desire of my Department that we should relegate the production of eggs to a lower category than they really deserve.

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Animal Feeding-Stuffs

Mr. De la Bere asked the Minister of Food whether, in view of the decrease in animal feeding-stuffs imported into this country in 1946 as compared with 1945, he will make a statement as to his plans and intentions for a substantial increase to be made available throughout 1947.

Dr. Summerskill: We intend to purchase increased quantities of feeding-stuffs during 1947, as intimated in my reply to the hon. Member for Lonsdale (Sir I. Fraser) on January 27th. It is not possible to announce details in advance.

WORLD'S POULTRY CONGRESS, 1948

The Danish Government have invited the World's Poultry Science Association to hold the eighth World's Poultry Congress in their country in 1948. The actual date has not yet been arranged, but it is understood that it will be held between the middle of July and the middle of August.—*Farmers' Weekly.*

Important Announcement

In common with other weekly periodicals, "The Veterinary Record" will not be published for two weeks (February 22nd, March 1st) following the production of this issue. Prolongation of the suspension beyond that period is possible and will be determined by the Government in due course.

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Fuel Crisis Stops Publication of Periodicals

"At Least Two Consecutive Issues"

Our readers will be fully aware of the circumstances which have necessitated the suddenly imposed prohibition of publication announced above, and we can only express our deep regret that, following the issue of this number of *The Veterinary Record*, members will be deprived of their professional journal for a period which yet remains to be determined, but which it is earnestly hoped will not exceed the two consecutive issues stipulated. We are also much concerned at the consequent delay in publication to which contributions to every section of the journal, also advertisements and secretarial notices, will thereby be subjected.

The disturbing news was broken to us and our contemporaries on Monday night, when the Central Office of Information stated that after discussion between the Ministry of Fuel and Power and the Periodical Proprietors' Association, while it was agreed that publication of all weekly periodicals and trade and technical papers dated up to Saturday, February 15th, might take place, after that date there would be a suspension of publication of at least two consecutive issues of all periodicals, irrespective of whether they were printed or published inside or outside the restricted areas or from what source they drew their paper supplies. The agreement thus applied to all periodicals and publications other than daily, evening, Sunday, and provincial newspapers. It also meant that after this Saturday no power might be used for the production of any such periodical.

A special notice to advertisers appears on page vi.

NOTES AND NEWS

Diary of Events

Feb. 15th.—Meeting of the Biochemical Society, London School of Hygiene, Keppel Street, W.C.1, 11 a.m.

Feb. 18th.—71st Annual Meeting of the Eastern Counties Division, N.V.M.A., at Norwich (Royal Hotel), 11.30 a.m.

Feb. 19th.—Meeting of the Section of Comparative Medicine, Royal Society of Medicine, at 1, Wimpole Street, W.1, 5 p.m.

Feb. 19th.—Meeting of the Association of Veterinary Teachers & Research Workers (Scottish Regional Group), Glasgow Veterinary College, 83, Buccleuch Street, 3 p.m.

Feb. 20th.—Meeting of the Society of Veterinary Practitioners, at York (Station Hotel), 2.30 p.m.

Feb. 20th.—Meeting of the Derbyshire Division, N.V.M.A., at the Midland Agricultural College, 2 p.m.

Feb. 21st.—Meeting of the Sussex Division, N.V.M.A., at Brighton (Old Ship Hotel), 2.30 p.m.

Feb. 22nd.—R.V.C. *v.* Royal (Dick) Veterinary College R.F.C., at the University of London Athletic Ground, Motspur Park, Surrey. K.O. 10.30 a.m.

Feb. 22nd.—Meeting of the Society of Women Veterinary Surgeons, at Liverpool (Veterinary Hospital, Pembroke Place), 2.15 p.m.

Feb. 22nd.—Meeting of the Nutrition Society, London School of Hygiene, Keppel Street, W.C.1, 11 a.m.

Feb. 26th.—Annual Meeting of the Scottish Metropolitan Division, N.V.M.A., at the Royal "Dick" Veterinary College, Edinburgh, 2.30 p.m.

Feb. 26th.—**Annual General Meeting of the South-Eastern Division, N.V.M.A., at Maidstone (Royal Star Hotel), 2.30 p.m.**
Feb. 28th.—**Glasgow V.C., Students' Representative Council Annual Ball, Cranston's, Royal Exchange Square, Glasgow, 7.30 p.m. to 1 a.m.**
Feb. 28th.—**Special Meeting, N.V.M.A. General Purposes and Finance Committee, 36, Gordon Square, W.C.1, 10 a.m.**
Mar. 5th.—**R.(D.)V.C. Students' Representative Council Annual Ball, New Locarno Ballroom, Slateford Road, Edinburgh, 8 p.m. to 1 a.m.**
Mar. 6th.—**Meeting of the Southern Counties Division, N.V.M.A., at Bournemouth, 2 p.m.**
Mar. 14th.—**Annual Reunion Dinner of the Holme Park Platoon, Home Guard, at the Galleon Restaurant, London Street, Reading, 5.30 for 6 p.m.**
Mar. 20th.—**Meeting of the Western Counties Division, N.V.M.A., at Exeter.**
May 23rd.—**R.V.C. Students' Union Society Annual Ball, Grosvenor House, Park Lane, W., 8 p.m. to 1 a.m.**

R.C.V.S. Council Meetings

A meeting of the Council of the Royal College of Veterinary Surgeons for registration purposes, Thursday and Friday, March 27th and 28th, 1947.

Quarterly Meetings of Council of the R.C.V.S., Wednesday, Thursday and Friday, April 9th, 10th and 11th, 1947.

July 28th—Aug. 1st.—N.V.M.A. Annual Congress in Edinburgh.

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The Tuberculin Test

We are informed by the Chief Veterinary Officer, Ministry of Agriculture and Fisheries, that the present double intradermal comparative test is shortly to be replaced by a single comparative intradermal test. We shall shortly be publishing a résumé of the experimental work which has led to this change.

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New Zealand Government Vacancies

In connection with the advertisement, regarding the above vacancies, appearing in this issue, we feel that the following points should be brought to the notice of intending applicants. We understand that these are new appointments, and it is satisfactory to note that the New Zealand Department of Agriculture now appreciates the necessity of enlisting the services of members of the profession in connection with poultry husbandry. It is not clear from this advertisement, however, what opportunities for advancement in the Service will be afforded appointees, and this question is being taken up with the New Zealand Government.

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N.V.M.A. Booklet on "The Husbandry and Diseases of Calves"

We tender our apologies to those members who, in consequence of the inclusion of the above booklet in last week's intimation of recently issued N.V.M.A. publications, have, as requested, written to advise the General Secretary of non-receipt. Through a misunderstanding, issue of this booklet by the printers has been delayed, but they assure us that it will be distributed to members with this number of *The Veterinary Record*.

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New Appointments to the Staff of the Ministry of Agriculture

As notified in recent issues, Council, N.V.M.A., has decided that no advertisements for appointments to the veterinary staff of the Animal Health Division of the Ministry of Agriculture shall be accepted for *The Veterinary Record* pending the settlement, in a manner satisfactory to the Association, of the question of the status and salaries of the Veterinary Officers of the Division.

In the meantime, any members of the profession who may, nevertheless, contemplate applying for such posts, should these be advertised elsewhere, are requested first to communicate with the General Secretary of the Association at 36, Gordon Square, London, W.C.1.

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The Supply of Distemper Prophylactics

Further to the announcements which appeared in our issues of January 18th and 25th, the Officers of the Association met representatives of Messrs. Burroughs Wellcome & Co. on January 30th and discussed the future of distemper prophylactic supplies. It appears that most of the production difficulties have been overcome but that the supply of animals essential to the production and testing of distemper prophylactics, while greatly improved, is still the limiting factor. So far as the future animal supply position can be judged and the demand of the profession assessed, it seems reasonably certain that Messrs. Burroughs Wellcome & Co. will, after March 15th when the present allocation period ends, be in a position to meet all the requirements of the profession.

In these circumstances the Officers of the Association pressed that future supplies be on a "supply on demand" basis and the firm have agreed that this system will operate after March 15th. After that date practitioners may, therefore, order distemper prophylactics in anticipation that their orders will be met by return post. While a guarantee cannot be given at present, when the production outlook is not completely satisfactory and the requirements of the profession in a free market can only be estimated, the prospects are good. A flood of orders at any one time might easily temporarily swamp production capacity or, even, the distribution arrangements. (All orders received will be dealt with in rotation.)

The profession will, at least, take some satisfaction in the fact that a free market can be tried out and the position which it creates will be kept under close review.

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PERSONAL

An Apology

Erroneous Announcement of Appointment.—We are informed by Mr. Neil H. Mathieson, M.R.C.V.S., of Larkhall, Lanarkshire, that the announcement reproduced from a lay paper in our issue of January 11th (p. 48), of his appointment by the County Council of Ross and Cromarty as veterinary officer for the Gairloch district of Wester Ross, is incorrect. Mr. Mathieson has no intention of relinquishing any of the practices, either in Larkhall, Motherwell or Hamilton, of which he is Principal, and we tender him our sincere apologies for the embarrassment caused him by the insertion of this erroneous announcement.

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Degree.—Mr. Robert Fraser Gordon, B.SC. (ABER.), M.R.C.V.S., of the Ministry of Agriculture's Veterinary Laboratory, Weybridge, has been awarded his D.Sc. by the University of Aberdeen for a thesis on "Avian Salmonellosis."

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Mention in Despatches.—Captain J. Helme, M.R.C.V.S., has recently been mentioned in despatches for gallant and distinguished services in Burma while serving with the R.A.V.C. Captain Helme has now resumed practice with Messrs. Steward and Rutter, veterinary surgeons, of Gwynne House, Hereford, with whom he was an assistant before joining the Forces.

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Births.—McLintock.—On February 4th, 1947, at 10, Hillside Road, Thorpe, Norwich, to Pauline, wife of John McLintock, M.R.C.V.S., a sister for David.

Pettett.—On December 22nd, 1946, at Beech Hill Nursing Home, Barnstaple, to Ina, wife of Dudley Pettett, M.R.C.V.S., a son (Richard), brother for Jillian and John.

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Forthcoming Marriage.—Pitman—John.—**The engagement is announced between Kenneth John Pitman, M.R.C.V.S., only son of Mr. and Mrs. G. T. Pitman, 20, Manor Road, Manselton, Swansea, and Mary, elder daughter of Mr. and Mrs. D. H. John, 18, Argyle Street, Pembroke Dock.**

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Marriage.—Morgan—Calder.—The marriage took place on January 18th, 1947, at Sandridge Parish Church, St. Albans, Hertfordshire, of Kenneth Urquhart Morgan, M.R.C.V.S., of Christchurch, Hampshire, to Barbara Joy Calder, of St. Albans.

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R.C.V.S. OBITUARY

Miller, Augustus, Turf Club, Cairo, Egypt. Graduated London, July 15th, 1897. Died January 20th, 1947; aged 71 years.

Mr. Augustus Miller, M.R.C.V.S.

After a prolonged illness Mr. Miller died on January 20th, 1947, in the Anglo-American Hospital at the age of 71. As a resident of Cairo for about 40 years he became well known to many veterinary officers both before and during the two World Wars. He was for 30 years with the Egyptian Government Veterinary Service and retired some 15 years ago but retained his interest in veterinary work by remaining Honorary Superintendent of the Cairo Branch of the R.S.P.C.A.

He was a keen amateur gardener of repute and took many prizes at the Cairo flower shows. Latterly he was in charge of the gardens of Gezira Club.

Mr. Miller was on the Classification Committee of the Jockey Club of Egypt and also a member of the Stewards Committee of Gezira Race Club. He had a sound knowledge of Arabs and their breeding.

Socially he entertained greatly during the war and his house was always full of people either staying or in transit.

He was buried in the Protestant Cemetery, Old Cairo, on January 21st, 1947, and the funeral was attended by many prominent European and Egyptian residents of Cairo.

J. J.

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INTER-COLLEGE 'SOCCER

Continuing the series of Inter-College matches so successfully inaugurated last season, the London 'Soccer Club were entertained by Liverpool on February 1st.

After a very enjoyable game, considering the atrocious ground conditions, Liverpool were the victors by three goals to two.

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Tuberculosis (Attested Herds) Scheme

STATEMENT SHOWING POSITION OF "ATTESTED HERDS" IN EACH COUNTY AS AT DECEMBER 31ST, 1946.

County	Total Cattle as on 4.6.46	Certificates of Attestation in force as at 31.12.46	Approximate Number of Cattle covered by Certificates of Attestation as at 31.12.46	Percentage of Attested Cattle to Total Cattle
Bedford	45,261	38	2,039	4·5
Berks	77,535	253	13,765	17·8
Buckingham	105,327	133	7,251	6·9
Cambridge	29,873	49	2,180	7·3
Isle of Ely	19,753	7	262	1·3
Chester	240,152	176	8,711	3·6
Cornwall	249,395	176	6,693	2·7
Cumberland	213,743	538	25,769	12·1
Derby	186,312	54	2,959	1·6
Devon	370,717	341	13,534	3·7
Dorset	134,402	267	13,781	10·3
Durham	115,913	183	7,028	6·1
Essex	122,189	279	12,946	10·6
Gloucester	176,003	196	10,949	6·2
Hampshire	137,970	297	14,076	10·2
Isle of Wight ..	20,217	17	761	3·8
Hereford	123,949	131	7,648	6·2
Hertford	57,048	129	8,260	14·5
Huntingdon	28,361	10	473	1·7
Kent	114,015	202	9,143	8·0
Lancaster	280,063	199	9,129	3·3
Leicester	156,465	76	3,921	2·5
Lincoln (Holland) ..	27,151	1	41	0·2
Lincoln (Kesteven) ..	68,054	8	319	0·5
Lincoln (Lindsey) ..	159,058	21	1,201	0·8
London	129	—	—	—
Middlesex	7,477	14	454	6·1
Norfolk	166,685	149	9,939	6·0
Northampton	143,645	44	2,635	1·8
Soke of Peterborough	7,093	—	—	—
Northumberland ..	192,051	160	7,622	4·0
Nottingham	109,818	47	2,129	1·9
Oxford	96,269	233	12,035	12·5
Rutland	20,435	8	889	4·4
Salop	261,528	207	11,540	4·4
Somerset	299,159	432	20,713	6·9
Stafford	231,152	68	2,435	1·1
Suffolk, East ..	76,987	59	3,084	4·0
Suffolk, West ..	35,015	54	3,178	9·1
Surrey	48,958	200	8,114	16·6
Sussex, East ..	96,208	159	7,184	7·2
Sussex, West ..	67,044	140	6,473	9·7
Warwick	144,096	101	6,218	4·3
Westmorland	94,057	378	17,677	18·8
Wilts	180,051	193	13,580	7·5
Worcester	92,685	67	3,586	3·9
Yorks, East Riding ..	125,648	18	672	0·5
Yorks, North Riding ..	237,172	347	11,449	4·8
Yorks, West Riding ..	340,074	246	10,788	3·2
ENGLAND ..	6,335,362	7,106	345,323	5·5
Anglesey	56,084	21	983	1·8
Brecon	45,041	156	4,834	10·7
Caernarvon	61,768	104	3,711	6·0
Cardigan	73,261	3,077	43,997	60·1
Carmarthen	131,629	3,645	70,703	53·7
Denbigh	98,253	326	8,205	8·4
Flint	58,571	43	1,853	3·2
Glamorgan	74,775	126	3,445	4·6
Merioneth	41,478	796	15,017	36·2
Monmouth	64,885	109	4,324	6·7
Montgomery	92,162	316	10,840	11·8
Pembroke	98,217	1,505	29,147	29·7
Radnor	35,440	47	1,260	3·6
WALES	931,164	10,271	198,319	21·3
ENGLAND & WALES	7,266,526	17,377	543,642	7·5
Aberdeen	204,491	83	7,705	3·77
Angus	56,952	66	5,894	10·35
Argyll	60,768	322	12,694	20·89
Ayr	142,911	1,743	106,736	74·68
Banff	50,737	20	1,598	3·15
Berwick	30,348	23	1,586	5·23
Bute	10,462	319	8,288	79·03
Caithness	24,574	130	1,866	7·59
Clackmannan	4,379	20	1,308	29·87
Dumfries	97,008	902	53,314	54·96
Dunbarton	16,438	124	7,050	42·89
East Lothian	14,364	16	1,766	12·29
Fife	57,318	127	9,790	17·08
Inverness	51,629	35	2,261	4·38
Kincardine	33,405	22	2,129	6·37
Kinross	7,779	28	1,728	22·21
Kirkcudbright	71,224	496	37,009	51·96
Lanark	91,239	851	48,588	53·25
Midlothian	23,403	101	5,506	23·53
Moray	27,954	31	2,773	9·92
Nairn	7,888	11	791	10·03
Orkney	43,927	153	3,254	7·41
Peebles	10,633	96	4,507	42·39
Perth	83,782	109	7,300	8·71
Renfrew	31,041	219	13,215	42·57
Ross and Cromarty ..	38,110	23	1,244	3·26
Roxburgh	32,350	25	1,546	4·79
Selkirk	5,954	19	687	11·54
Stirling	40,319	217	12,610	31·28
Sutherland	9,948	6	302	3·04
West Lothian	14,196	67	3,710	26·13
Wigtown	68,143	328	28,149	41·31
Zetland	8,339	1,246	4,516	54·16
TOTAL SCOTLAND	1,472,013	7,978	401,400	27·27
TOTAL GT. BRITAIN	8,738,539	25,355	945,042	10·82

* * * * *

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

FOOT-AND-MOUTH DISEASE:

Essex.—Wick Farm, Southchurch, Southend-on-Sea, Essex (February 5th).

Leics.—Moors Farm, Hathern, Loughborough, Leicestershire (February 6th); Moor Farm, Colcorton, Leicester (February 7th).

SWINE FEVER:

Northumberland.—Saugh House Field, New Northumberland Dock, Dock Road, Perey Main, Tynemouth, Northumberland (February 5th).

Surrey.—The Cottage, 57, Beulah Hill, Upper Norwood, S.E.19.

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LEGAL NOTES

"Cattle" Includes Pigs.—Last year we recorded the legal decision that "sheep *are* cattle," and more recently, in a revenue case which raised the question whether or not the word "cattle" in Rule 4 of Case III of Schedule D to the Income Tax Act, 1918, included pigs, Mr. Justice Atkinson held that cattle did include pigs. In the course of his judgment he said that, when the present translation of the Bible was made, cattle included all kinds of animals, and that Adam gave names to all cattle. Although, according to dictionaries, the present tendency was to restrict the term "cattle" to the bovine genus, it seemed clear that pigs could properly be included.

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"DICK" COLLEGE DRAMATIC SOCIETY

We are glad to have such evidence of the reviving cultural activities of our Colleges as is provided by a note recently received from the Hon. Secretary of the Royal (Dick) Veterinary College Dramatic Society. "This is a very young Society," she writes, "and so far we have produced two plays, our first being 'The Bird in Hand' by Drinkwater, while last November we produced 'The Ghost Train' by Arnold Ridley. This term we are proposing to give a performance of 'The Late Christopher Bean' by Emlyn Williams—and the proceeds will go to the Victoria Veterinary Benevolent Fund.

"We thought these facts would interest you, and maybe other Colleges as well, as we find that the Society is a great success here."

An admirable example, indeed—and more particularly the happy decision to devote the "returns" of the forthcoming performance to the ever-needy resources of the "Victoria" Fund.

NATIONAL AGRICULTURAL ADVISORY SERVICE

The Ministry of Agriculture and Fisheries announces the following appointments:—

County Advisory Officers

(Except where otherwise stated, the County Advisory Officer is also the Executive Officer of the County War Agricultural Executive Committee.)

Northern Province

Cumberland—J. H. Faulder, B.Sc., N.D.D.
Durham—R. Bruce, B.Sc.
Northumberland—I. C. Jack, B.Sc., PH.D.
Westmorland—D. T. Adam, B.Sc., N.D.A., F.S.I.

Yorks. and Lancs. Province

Lancashire—T. C. Creyke, N.D.A., N.D.D.
Yorkshire (East Riding)—R. B. Ferro, N.D.A.
(North Riding)—D. S. Hendrie, B.Sc., N.D.A., N.D.D.
(West Riding)—J. Gibbons, M.Sc. (Executive Officer: W. P. Richardson, O.B.E., F.L.A.S.)

West Midland Province

Cheshire—W. A. C. Carr, N.D.A.
Hereford—E. L. Jones, M.Sc., N.D.A. (Executive Officer: Grevile Phillips, F.A.I., M.INST.R.A., F.L.A.S.)
Salop—J. W. Reid, N.D.A.
Stafford—H. E. Wells, N.D.A., N.D.D.
Warwick—W. E. Gelling, B.Sc., N.D.A.
Worcester—H. N. Jacobs, N.D.A., P.A.S.I.

East Midland Province

Derby—A. D. R. Walbank, N.D.A.
Leicester—R. T. J. Fleming, N.D.D.
Lincs. (Kesteven)—G. B. Wells, N.D.A., N.D.D.
(Lindsey)—A. Mann, B.Sc.
Northampton—K. J. Allright, B.Sc., N.D.A. (Executive Officer: R. George, F.L.A.S.)
Nottingham—N. F. McCann, B.Sc., N.D.A.
Rutland—I. P. McEwen, B.Sc.

Eastern Province

Bedford—J. W. Dallas, M.Sc.
Cambridge—E. T. Sykes, M.A.
Essex—L. D. C. McLees, B.Sc., N.D.A., N.D.D.
Hertford—R. Line, B.Sc., N.D.A.
Huntingdon—M. Ward, N.D.A.
Isle of Ely—P. E. Cross, N.D.H. (Executive Officer: R. T. Wesley, O.B.E.)
Lincs. (Holland)—J. C. Wallace, M.C.
Norfolk—J. C. Mann, M.A., A.I.C.
Soke of Peterborough—W. A. Scriven, N.D.A., N.D.D.
Suffolk (East)—P. J. O. Trist, B.A.
(West)—R. Sayce, B.Sc., N.D.A.

South-Eastern Province

Berkshire—S. L. Huthnance, B.Sc., N.D.D.
Buckingham—V. P. Walley, N.D.A., P.A.S.I.
Hampshire—T. W. Williamson, B.Sc., N.D.D.
Isle of Wight—B. J. Fricker, N.D.A., N.D.D.
Kent—R. Duncan, N.D.H.
Middlesex—N. J. Wood, B.A.
Oxford—Vacancy. (Executive Officer: B. J. S. McFie, F.S.I., A.F.A.S.)
Surrey—S. J. Travers, M.B.E., N.D.A.
Sussex (East)—W. S. Gibson, B.Sc.
(West)—E. A. Bartlett, N.D.A. (Executive Officer: W. Lawson, M.B.E., N.D.A., N.D.D.)

South-Western Province

Cornwall—A. Gregg, B.Sc., N.D.A. (Executive Officer: J. Wilson, O.B.E., F.L.A.S.)
Devon—W. Williams, M.Sc.
Dorset—T. R. Ferris, O.B.E., M.Sc., N.D.D.
Gloucester—J. R. Clapham, B.Sc., N.D.A., N.D.D.
Isles of Scilly—F. W. Shepherd, N.D.H.
Somerset—D. C. Bower, N.D.A., N.D.D.
Wiltshire—W. J. Brimacombe, M.Sc.

Welsh Province

Anglesey—E. Jones, B.Sc.
Brecon—J. Davies, B.Sc.
Caernarvon—H. E. Roberts, B.Sc.
Cardigan—D. J. Morgan, B.Sc.
Carmarthen—D. S. Davies, B.Sc.
Denbigh—T. I. Jones, N.D.D.
Flint—J. H. Humphreys, M.Sc. (Executive Officer: H. Broad.)
Glamorgan—E. L. Harry, O.B.E., M.Sc.
Merioneth—E. E. Williams, N.D.A.
Monmouth—J. H. Morgan, B.Sc.
Montgomery—E. David, B.Sc.
Pembroke—W. H. Jones, M.Sc.
Radnor—H. E. Evans, B.Sc.

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REUNION DINNER

The Annual Reunion Dinner of the Holme Park Platoon, Home Guard, will be held on Friday, March 14th, 1947, at 5.30 for 6 p.m., at the Galleon Restaurant, London Street, Reading, price 5s. R.A.V.C. personnel past and present are particularly invited to attend. Hotel accommodation can be arranged if requested.

All intending to be present, who have not already given notification, should communicate with C. W. Ottaway, Zoological Laboratory, Downing Street, Cambridge, by Saturday, March 1st, at the latest.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents should write as briefly as possible and letters should not exceed 350 words.

PROFESSIONAL STATUS: REMUNERATION OF THE ROYAL ARMY VETERINARY CORPS

Sir,—As an ex-R.A.V.C. Officer I would like to comment on the unsatisfactory reply of the Army Council to Brigadier Mosley published in your issue of January 25th, 1947.

1. The revised rate of pay for a Captain, R.A.V.C., during the first two years in that rank is *the lowest rate paid to a Captain in the whole Army*. Considering that for the last two or three years the majority of R.A.V.C. officers have been in that category, it follows that the Corps as a whole is not being paid as generously as the Army Council claims.

2. One would imagine from the Army Council's reply that the R.A.V.C. is a non-combatant Corps. In S.E.A.C., and perhaps elsewhere, orders were issued to contradict that idea. R.A.V.C. officers and their units were combatants.

3. In precisely what respects does the responsibility of an R.A.V.C. officer fall short of that of any other officer? Experience in the latest world war has shown that it is, in fact, often far greater.

In countless cases a veterinary officer of junior rank, with a few weeks' or months' service, and without O.C.T.U. training, has found himself in command of a veterinary unit. In this respect the veterinary officer was responsible for everything for which an officer can conceivably be held responsible, *e.g.*, men—their discipline, training, fitness, welfare, etc.; stores, vehicles, equipment, arms, etc.; and if he was in the field, responsibility for his unit's part in the scheme of operations, to the extent of commanding it in action against the enemy (as happened with several veterinary units in Burma.) Thus I maintain the Army Council has no justification for its yardstick measurement of a veterinary officer's responsibility.

Having drawn the attention of the profession to only three glaring inaccuracies in the Army Council's reply, and having refrained through lack of space from commenting on the still unexplained difference in pay as compared with medical and dental officers, or on comparative pays when qualification pay is considered, I appeal to the profession as a whole to reject the Army Council's reply, and to take the matter up again.

Yours faithfully,

M. M. MacKinnon.

Wood House,
The Ridgeway,
Tonbridge, Kent.
January 30th, 1947.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Parasitic Mange*	Sheep Scab	Swine Fever
Jan. 1st to 15th, 1947 ...	1	20	—	4	2
Corresponding period in—					
1946 ...	9	4	1	3	19
1945 ...	9	8	1	7	46
1944 ...	12	1	1	22	21

Note.—The figures for the current year are approximate only.
* Excluding outbreaks in Army Horses.

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period October 1st to December 31st, 1946, was 1,832.

N.V.M.A ANNUAL CONGRESS, 1947

July 28th - August 1st (inclusive)

LIST OF HOTELS, ETC., IN EDINBURGH AND SURROUNDING DISTRICTS

It is desirable that applications for accommodation should be made as early as possible as hotels and boarding-houses are already booking for next summer.

A small number of rooms has been reserved for the Congress week in the following Hotels: Adelphi, Caledonian, George, North British, Royal British, and Roxburghe. Members desirous of staying at any of these should communicate direct with the Local Secretary, Professor A. Robertson, Royal (Dick) Veterinary College, Edinburgh, 9.

Name and Address of Hotel	Terms per Week	Terms per Day
Licensed		
Braid Hills Hotel, Edinburgh	7–8 gns.	—
Caledonian Hotel, Princes St., Edinburgh	9½–12½ gns.	—
Cockburn Hotel, Edinburgh	6 gns.	—
George Hotel, 21, George St., Edinburgh	—	£1/0/6–£1/2/6
Harp Hotel, Corstorphine, Edinburgh	6 gns.	—
North British Station Hotel, Princes St., Edinburgh	—	25/– – 28/6
Queen Hotel, Edinburgh	7–8 gns.	—
Roxburghe Hotel, Charlotte Sq., Edinburgh	—	27/–
Royal British Hotel, 20, Princes St., Edinburgh	8 gns.	—
•Rutland Hotel, 3, Rutland St., Edinburgh	—	12/6–18/6
Unlicensed		
Abercromby Hotel, Edinburgh	4½–5½ gns.	—
Afton Hotel, Grosvenor Cres., Edinburgh	£6/9/6–7 gns.	—
Alexander Hotel, 1, Hunter Sq., Edinburgh	£3/10–£4	—
Alison Hotel, 1, Melville Cres., Edinburgh 3	5–8 gns.	—
Annorma House Hotel, 8, Nile Grove, Edinburgh 10	6 gns.	—
Bellevue Private Hotel, 13, Inverleith Row, Edinburgh 4	4–5 gns.	—
Blair Atholl Hotel, 10, Grosvenor Sq., Edinburgh 12	4½–5½ gns.	—
Carahane Hotel, 1, Peel Ter., Newington, Edinburgh 9	7–10 gns.	—
Carlton Private Hotel, Edinburgh	5 gns.	—
Chimes Hotel, 3, Royal Ter., Edinburgh	6 gns.	—
Clifton Hotel, 53, Promenade, Portobello, Edinburgh	4½–6 gns.	—
Crown Hotel, Torphichen St., Edinburgh	5 gns.	—
Darlings Regent Hotel, 21, Waterloo Pl., Edinburgh	£7/14	—
Donmaree Hotel, 21, Mayfield Gardens, Newington, Edinburgh 9	7–8 gns.	—
Edward Hotel, 57, Gt. King St., Edinburgh	5 gns.	—
Ettrick Private Hotel, Edinburgh	5–6 gns.	—
Gloucester Hotel, Gloucester Pl., Edinburgh	4½–5½ gns.	—
Gordon Hotel, 7, Royal Circus, Edinburgh 3	3½–6 gns.	—
Grange Hotel, 10, Chalmers Cres., Edinburgh	£3/10–4 gns.	—
Green's Hotel, Eglinton Cres., Edinburgh	5½–7 gns.	—
Halcyon Hotel, 8, Royal Ter., Edinburgh	5½ gns.	—
Haymarket Hotel, 1, Coates Gdns., Edinburgh	4 gns.	—
Holme Hotel, 12, Royal Circus, Edinburgh 3	3½–5 gns.	—
Iona Private Hotel, Strathearn Pl., Edinburgh	5 gns.	—
Janette Hotel, 9, Royal Ter., Edinburgh	5 gns.	—
Lea Private Hotel, 69, Leamington Ter., Edinburgh	5 gns.	—
Leamington Hotel, Leamington Ter., Edinburgh 10	7 gns.–£8/15	—
†Magdala Hotel, 4, Magdala Cres., Edinburgh 12	—	7/6–8/6
Maitland Hotel, 33, Shandwick Pl., Edinburgh	5–6 gns.	—
Mayfield Private Hotel, 15, Mayfield Gdns., Edinburgh 9	5½–6 gns.	—
Melrose Private Hotel, 47, Minto St., Edinburgh	4½ gns.	—
Menzies Hotel, 26, Royal Ter., Edinburgh	5 gns.	—
Palmerston Private Hotel, 56, Palmerston Pl., Edinburgh	—	13/6–15/–
Pirie's Hotel, 6, Coates Gdns., Edinburgh	5 gns.	—
Regent Private Hotel, 6, Regent Ter., Edinburgh 7	3½–4½ gns.	—
Rosslyn Private Hotel, 9, Palmerston Pl., Edinburgh	3½–5 gns.	—
Royal Stuart Hotel, 18, Abercromby Pl., Edinburgh	4½–7 gns.	—
Shelbourne Hotel, Edinburgh	£6/9/6	
St. Alban's Hotel, 9, Rutland Sq., Edinburgh	£4/14–£5/10	
St. Andrew Hotel, 8, South St., Andrew St., Edinburgh 2	£6/2/6–7 gns.	—
Suttie's Hotel, Edinburgh	6 gns.	—
Terrace Hotel, Edinburgh	5 gns.	—
Varrich House Private Hotel, 7, Church Hill, Edinburgh 10	5–6 gns.	—
Boarding Houses		
Greenhill House, 12, Strathearn Pl., Edinburgh	4–5 gns.	—
Learmonth Guest House, 11, South Learmonth Gdns., Edinburgh	—	12/6
Mayflower Board Residence, 15, Glenorchy Ter., Edinburgh 9	—	11/– – 12/–
Richmond House, 20, Leopold Pl., Edinburgh	—	9/– – 10/6
Springbank, 22, Bath St., Portobello, Edinburgh	£3/10	—
Tigh-na-Bruaich, 57, Orchardhead Rd., Liberton, Edinburgh 9	5 gns.	—
West Colinton Guest House, Colinton, Edinburgh 13	5 gns.	—
Hotels — EAST LOTHIAN		
Goblin Ha' Hotel, Gifford	5 gns.	—
Bissets Hotel, Gullane	7 gns.	—
Linga Hotel, Gullane	6–7 gns.	—
Queen's Hotel, Gullane	Opening in the Spring—Terms not yet quoted	
Ardrulin Private Hotel, Clifford Rd., N. Berwick	7 gns.	—
County Hotel, High St., N. Berwick	5 gns.	—
Dunedin Lodge, N. Berwick	£6	—
King's Knoll Hotel, Clifford Rd., N. Berwick	7 gns.	—
Marine Hotel, N. Berwick	£9/2/6–£12/12/6	
Royal Hotel, Station Rd., N. Berwick	7–8 gns.	—
Castle Hotel, Dirleton	5 gns.	—
Craig-en-Gelt Hotel, Dunbar	6 gns.	—
George Hotel, Haddington	4 gns.	—
MIDLOTHIAN		
Juniper Lea, Blackshiels	5 gns.	—
Harrow Hotel, Dalkeith	6–7 gns.	—
Orford House Private Hotel, Dalkeith	—	15/– – 18/–
‡Melville Castle, Lasswade	5–7 gns.	—
Royal Hotel, Stow	£4/10	—
WEST LOTHIAN		
Cramond Bridge Hotel, Cramond	4 gns.	—
Hawes Inn, South Queensferry	5½–6 gns.	—
Queensferry Arms Hotel, S. Queensferry	£4/7/6	—
Staghead Hotel, South Queensferry	£7–8 gns.	—
Roadhouse Hotel, Near Whitburn	5½ gns.	—
PEEBLES-SHIRE		
Traquair Arms Hotel, Innerleithen	5–6 gns.	—
Green Tree Hotel, Peebles	£5	—
Tontine Hotel, High St., Peebles	5 gns.	—
Waverley Hotel, High St., Peebles	5 gns.	—
Cross Keys Hotel, Peebles	5 gns.	—
Minden Hotel, Innerleithen Rd., Peebles	5 gns.	—
Romanno Bridge Hotel, Romanno Bridge	£4/10	—

• Meals extra. † Bed and breakfast only. ‡ Opening Mar. 1st.